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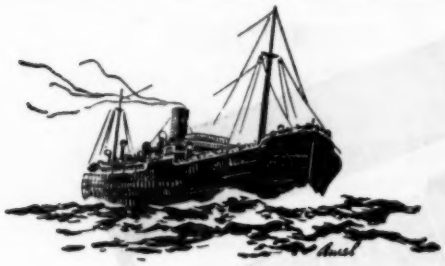

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Journal of the Indian Medical Association

VOL. XXI, NO. 4 CALCUTTA JANUARY, 1952

I.M.A. WORKING COMMITTEE

PROCEEDINGS OF THE 41ST MEETING

[SUBJECT TO CONFIRMATION]

The 41st Meeting of the Working Committe of the Indian Medical Association was held at Gauhati on the 14th and 15th October, 1951, with Dr. T. N. Banerjee in the Chair.

MEMBERS PRESENT: (a) *Ex-Officio*: 1. Dr. T. N. Banerjee of Patna (President), 2. Dr. Chamanlal M. Mehta of Bombay (Immediate Past President), 3. Dr. A. K. Sen of Calcutta (Senior Vice-President), 4. Dr. S. C. Sen of Delhi (Hony. General Secretary), 5. Dr. A. P. Mittra of Delhi (Honorary Joint Secretary), 6. Dr. D. Prosad of Patna (Honorary Joint Secretary), and Dr. A. D. Mukharji of Calcutta (Editor of J.I.M.A.).

(b) *Representatives from the Provincial Branches*: 8. Dr. A. C. Ukil, 9. Dr. P. K. Chatterjee, 10. Dr. J. Mojumdar and 11. Dr. M. K. Roychaudhury of the Bengal Provincial Branch; 12. Dr. S. Samaddar and 13. Dr. D. D. Prosad of the Bihar Provincial Branch; 14. Dr. T. J. Lalvani and 15. Dr. (Miss) V. S. Kalewar of the Bombay Provincial Branch; 16. Dr. A. P. Shukla and 17. Dr. B. G. Mehta of the Gujarat and Saurashtra Provincial Branch; 18. Dr. R. A. Bhagwat of the Madhya Bharat Provincial Branch; 19. Dr. B. K. Vinchure of the Madhya Pradesh Provincial Branch; 20. Dr. B. V. Mulay and 21. Dr. R. C. Thanawala of the Maharashtra and Karnatak Provincial Branch; 22. Dr. Ram Rakha Dheer of the Punjab Provincial (State) Branch; 23. Dr. C. O. Karunakaran of the Travancore-Cochin Provincial Branch; 24. Dr. H. Hukku and 25. Dr. S. N. Saxena of the Uttar Pradesh Provincial (State) Branch.

(c) *Co-opted*: 26. Dr. Bhubaneswar Baruah of Gauhati; 27. Dr. H.C. Baruah of Shillong; 28. Dr. P. K. Guha of Calcutta; 29. Dr. A. K. Guha and 30. Dr. P. N. Sinha of Patna.

(d) *Invited*: 31. Dr. P. C. Duarah and 32. Dr. N. K. Sharma of Gauhati.

Item 1: *CONDOLENCE RESOLUTION.*

The following condolence resolution was unanimously passed, all members standing and observing silence for 1 minute:—

"This meeting of the Working Committee of the Indian Medical Association places on record its deep sense of sorrow at the sad demise of the following members of the Association and conveys its heartfelt sympathy to the members of the bereaved families:—

1. Dr. Amar Nath Goyle (Amritsar), 2. Dr. S. P. Chaudhury (Bettiah), 3. Dr. S. M. Farid (Darbhanga), 4. Major N. D. Banerjee (Kanpur), 5. Dr. S. B. Patankar (Sholapur), 6. Dr. V. B. Kale (Sholapur), 7. Dr. Sadiq Ali (Sultanpur), 8. Dr. P. S. Nannadiar (Nilgris), 9. Dr. I. H. Rizvi (Lucknow), 10. Dr. S. G. Shukla (Jabalpur) and 11. Dr. P. Gupte (Muzaffarpur)."

Item 2: *MESSAGES OF INABILITY TO ATTEND.*

Messages were received from the following members for their inability to attend this meeting:—1. Dr. H. R. Dawar of Delhi (Hony. Treasurer), 2. Dr. R. Sinha of Calcutta (Hony. Joint Secretary); 3. Dr. B. T. Rao of Andhra Provincial Branch; 4. Dr. G. K. Ghosh of Bihar Provincial Branch; 5. Dr. C. S. Thakkar and 6. Dr. B. R. Nayen of the Bombay Provincial Branch; 7. Dr. J. N. Bahadur of Delhi Provincial Branch; 8. Dr. C. P. Bhatt and 9. Dr. P. R. Trivedi of Gujarat & Saurashtra Provincial Branch; 10. Dr. G. V. Joshi of Maharashtra and Karnatak Provincial Branch; 11. Dr. D. G. Ojha of Rajputana Provincial Branch; 12. Dr. U. Krishna Rao and 13. Dr. P. A. S. Raghavan of the South Indian Provincial Branch and 14. Capt. H. N. Shivapuri of the Uttar Pradesh Provincial (State) Branch.

Item 3: *CONFIRMATION OF THE PROCEEDINGS OF THE LAST MEETING OF THE WORKING COMMITTEE.*

The Proceedings of the 40th meeting of the Working Committee held at Hyderabad on the 7th and 8th July 1951, as circulated, were confirmed, with minor corrections.

Item 4: *BUSINESS ARISING OUT OF THE PROCEEDINGS OF THE LAST MEETING OF THE WORKING COMMITTEE.*

Before this item was taken up, it was suggested by Dr. A. C Ukil and some other members that, in future, sub-items under this Item should be placed at the meeting and discussed with reference to the items of business transacted at the last meeting. As this procedure would be helpful to the members, the suggestion was accepted by the Hony. General Secretary.

4(1): *Pay, status etc. of Medical Officers*: Dr. S. C. Sen, the Hony. General Secretary, informed the house that the special sub-committee could not yet finalise the formulation of a comprehensive scheme regarding the pay, prospects and status of the railway and other medical officers. This was noted.

4(2): *Essential drugs and appliances*: The Hony. General Secretary informed the house that the list of essential drugs, instruments and appliances for hearing aid etc., received from Calcutta and Madras were sent to the Bombay Provincial Branch and their report on the matter was awaited. This was noted.

4(3): *Fee scales for examination of Life Insurance cases*: The Hony. General Secretary informed the house that Dr. Chamanlal Mehta was expected to place before the house some concrete proposals at the next meeting of the Working Committee. It was noted.

4(4): *Mutual affiliation of I.M.A. with B.M.A.*: The Hony. General Secretary read out a letter dated 20-7-51 of

the British Medical Association clarifying the interpretation of certain clauses, regarding the mutual affiliation of the I.M.A. with the B.M.A.

Dr. B. Baruah and Dr. H. C. Baruah requested the Hony. General Secretary to see that the Assam Branch of the British Medical Association co-operated with the Assam Provincial Branch of the Indian Medical Association in matters concerning the Indian doctors serving in the British-managed tea gardens of Assam. A general discussion followed in which Dr. S. C. Sen, Dr. A. C. Ukil and other members took part. The matters discussed were noted.

4(5): *Land for I.M.A. Building at Delhi*: The Hony. General Secretary informed the house regarding the steps taken by him for acquiring of a plot of land for the central office of the Indian Medical Association. In this connection, Dr. A. C. Ukil informed the house that the National Institute of Sciences of India could manage to acquire a plot of land at Delhi with the assistance of the Government. Dr. P. K. Guha reminded the Hony. General Secretary that while he was the Hony. General Secretary, he had sent an application to the National Institute of Sciences of India for their assistance to the I.M.A. in securing a liberal grant from the Government of India for the building, library, publications etc., of the I.M.A. After a general discussion in which the President, Dr. A. C. Ukil, Dr. S. Samaddar and other members took part, the Hony. General Secretary assured the house that he would explore all possible avenues and would also contact the National Institute of Sciences of India in this matter.

The Hony. General Secretary also informed the house that Rs. 4,608/6/- had already been collected and a further sum of Rs. 4,235/- was promised as donation to the I.M.A.—Building Fund. He suggested, in this connection, that every member of the Indian Medical Association should be requested to contribute at least Rs. 50/- in one instalment or in easy instalments of Rs. 5/- per month, for this purpose, so that 5 lakhs of rupees could be raised for the building fund. Dr. T. N. Banerjee, the President assured the house that he would issue an appeal at once and it would be printed as a slip and pasted on the cover of the November '51 issue of the Journal. Dr. A. D. Mukharji requested the President to include matters re: "Your Health" in this appeal so that some more members could be enlisted as subscribers of "Your Health." Dr. Mukharji's suggestion was also accepted by the President.

4(6): *Disposal of the I.M.A. Benevolent Fund*: The Hony. General Secretary read out the proceedings of the Special Committee appointed by the Working Committee for the disposal of the Benevolent Fund of the I.M.A. He also said that that the fund was now being closed. The actions of the Committee and the Hony. General Secretary were approved.

4(7): *Training of village Vaids by the Government*: Dr. B. V. Mulay informed the house that the Government scheme of training of the village vaids was not in the best interests of the people and the Travancore-Cochin Provincial Branch was right in its protest.

After a general discussion in which Dr. B. V. Mulay, Dr. C. O. Karunakaran, Dr. R. A. Bhagwat, Dr. B. K. Vinchure, Dr. C. M. Mehta, Dr. A. C. Ukil, Dr. P. K. Guha and others took part, the Hony. General Secretary was requested to take necessary action in this matter.

4(8): *New Rules of the I.M.A.*: The Hony. General Secretary informed the house that he had sent a set of skeleton draft rules and read out the Ad-interim report of the Ad-hoc Committee for re-drafting the rules of the Indian Medical Association. These were noted.

4(9): *Standard of Medical Education*: The Hony. General Secretary informed the house that the Medical Education and Health Services Sub-Committee could not yet meet and submit its report on the standard of medical education in India. This was noted.

4(10): *Position re. Resident House Appointments Abroad*: The Hony. General Secretary informed the house, with regret, that a few post-graduate medical students sent abroad through the Indian Medical Association as resident house staff in foreign hospitals resigned their jobs, for various reasons. He also stated that no further offers regarding resident house appointments abroad had been received by him so far. These were noted.

4(11): *Import and Distribution of Essential Drugs*: The Hony. General Secretary stated that the Educational and Scientific Standing Committee appointed by the Working Committee could not yet finalise the scheme of import and distribution of essential drugs like Penicillin, Streptomycin, Chloromycetin and Sulphonamides. This was noted.

4(12): *System of appointment of Hony. Medical Officers*: The Hony. General Secretary informed the house that a joint meeting of the Educational and Scientific Standing Committee and the Medical Education and Health Services Sub-Committee was held but their scheme regarding the system of Honorary Medical Officers for teaching and other hospitals could not yet be finalised. This was noted.

In this connection, Dr. P. K. Guha requested the Hony. General Secretary to enquire from the State Governments whether Hony. Medical Officers attached to Government hospitals were treated as Government servants and as such were subject to the conduct and leave rules etc., of the State Governments. Dr. Guha also requested the Hony. General Secretary to try to clarify the status of the Hony. Visiting Physicians, Surgeons and Specialists of teaching hospitals under the State Governments, in relation to Presidency Surgeons, Civil Surgeons, Gazetted Medical Officers etc. The Hony. General Secretary promised to take up the matter.

4(13): *T. A. Bill of Lt.-Col. T. S. Shastry*: The Hony. General Secretary informed the house that as per direction of the President, Lt.-Col. T. S. Shastry's (of Madras) T. A. Bill for attending the Working Committee meeting held at Delhi on the 24th and 25th March, 1951 was paid. The opinion of the Auditor of the I.M.A. in this connection, was also read out. The actions of the President and the Hony. General Secretary in this matter were approved.

4(14) (a): *Commonwealth Medical Conference in India in 1952*: The Hony. General Secretary informed the house that as the representative of the I.M.A. to the Conference, he had invited the members of the Conference to hold their next meeting in India in February or March 1952, at Ahmedabad, Bombay or Delhi. Dr. Sen's suggestion was approved and he was authorised to proceed further in the matter, regarding date and other arrangements.

In this connection, Dr. S. C. Sen informed the house that it might not be possible for the Commonwealth Medical Conference to hold their next sitting at Ahmedabad during our Conference. In that case, he added, that the Conference could be invited at Delhi, Bombay or Calcutta. Dr. Sen pointed out that the I.M.A. would have to elect a member to preside over the session of the Commonwealth Medical

Conference to be held in India. Hon'ble Dr. B. C. Roy, the Seniorm[ost] Past President of the Association was unanimously elected f[or] this purpose. In case Hon'ble Dr. Roy could not preside, it was also decided to request the Hon'ble Dr. Jivraj Mehta, the next seniormost Past President of the I.M.A. to preside over the Conference.

The Hony. General Secretary also informed the house that the Association would have to incur the necessary expenditure for the Commonwealth Conference. This was noted and he was requested to proceed in this matter.

4(14) (b): *W. M. A. Council Meeting in India in 1952 or 1953*: The Hony. General Secretary informed the house that he had invited the W.M.A. to hold a meeting of its Council in India in 1952 or 1953. The suggestion was approved and Dr. Sen was requested to proceed further in the matter.

4(15): *Lay Health Journal—"YOUR HEALTH"*: The Hony. General Secretary informed the house that "Your Health," the new health journal of the I.M.A. for the lay public, would be published from January 1952 from the office of the Journal of the I.M.A.

Dr. A. D. Mukherji who was entrusted by the Working Committee at its last meeting to prepare a scheme for its publication from January 1952, placed before the house a few copies of the "dummy" of "YOUR HEALTH" prepared by him, with the assistance of Dr. Guha. Dr. Mukherji suggested that Rs. 5,000/- should be set apart, now, for "Your Health" from the funds of the Journal of the I.M.A., so that the first issue of "Your Health" could be published in due time. He also reminded the house that the new health journal might have to be published during the first few months at a loss. On a question from Dr. Mulay, Dr. S. C. Sen, Hony. General Secretary said that after the scheme of publication of "Your Health" in English from Calcutta became successful and profitable, the health journal could be published in Hindi, Bengali and other regional and State languages by the Provincial Branches of the I.M.A., if they so desired.

The Hony. General Secretary then reminded the house that they would have to elect an Editor for "Your Health" and would have to pass a resolution regarding its banking account. A general discussion then followed in which Dr. Mehta, Dr. Mulay, Dr. S. C. Sen, Dr. A. K. Sen, Dr. Karunakaran, Dr. Ukil, Dr. Lalvani, Dr. Mukharji, Dr. Chatterjee, Dr. Mojumdar, Dr. Roychaudhury and others took part. Dr. M. K. Roychaudhury suggested that the present Journal Committee of the I.M.A. should be entrusted with the work of publication of "YOUR HEALTH."

The following resolutions were moved by Dr. P. K. Chatterjee and seconded by Dr. J. Mojumdar:—

"Resolved that the 'dummy' of the lay health journal —"YOUR HEALTH"—as presented by the Journal Committee be approved. The Journal Committee be requested to proceed to publish the journal, monthly, starting from January next.

"Further resolved that Dr. A. D. Mukherji be elected as the Editor of the lay Journal "YOUR HEALTH" and he be entrusted with both the editorial and business work of the new journal."

"Further resolved that Dr. P. K. Guha be elected as the Assistant Editor of "Your Health" to assist the Editor in both editorial and business matters.

"Further resolved that the present Journal Committee be given the same powers in respect to "Your Health" as is at present provided for in the rules in respect of the Journal of the Indian Medical Association."

"Further resolved that a separate account be opened in the name of "YOUR HEALTH" with the United Bank of India Ltd., New Market Branch, Calcutta, and a sum Rs. 5,000/- be transferred from the present journal funds to the new account as a working fund for "Your Health."

"Further resolved that the new banking account be operated jointly by any two of the following: (1) Editor (2) Hony. Joint Secretary of the I.M.A. at Calcutta, and the (3) Assistant Editor."

The following set of resolutions were, then, moved by Dr. Chamanlal Mehta and seconded by Dr. S. C. Sen:—

"Resolved that a lay health journal "Your Health" be published by the Association from January, 1952. A special committee consisting of Dr. A. K. Sen, Senior Vice-President at Calcutta, Dr. R. Sinha, the Hony. Joint Secretary at Calcutta Dr. A. D. Mukharji and Dr. P. K. Guha of Calcutta will conduct the affairs of the Journal with Dr. A. D. Mukherji as the Editor and Dr. P. K. Guha as the Assistant Editor."

"Resolved further that a new bank account be opened at the New Market Branch of the United Bank of India with Rs. 5,000/- transferred from the Journal Account. This new bank account will be operated jointly by any two of the four members mentioned above."

"Further resolved that this Committee will work for one year from the 15th of October, 1951."

The two resolutions were put to vote after a detailed discussion. 5 members voted for and 16 members against Dr. P. K. Chatterjee's resolutions, Dr. A. D. Mukharji and Dr. P. K. Guha remaining neutral. 15 members voted for and 4 against Dr. Mehta's resolutions, Dr. A. D. Mukharji and Dr. P. K. Guha again remaining neutral. Dr. Chamanlal Mehta's resolutions were declared as duly passed.

4(16): *Notification of Coal Mines Welfare Board*: The Hony. General Secretary read out the relevant correspondence re: a notification of the Coal Mines Welfare Board of the Government of India, Ministry of Labour. He also gave an account to the house regarding further details received from the Sitarampur Branch. These were noted and the Hony. General Secretary was requested to take necessary steps in the matter.

4(17): *Non-recognition of L.M.S. (Non. F. Sc.) diploma of Punjab State Faculty*: The Hony. General Secretary informed the house that a copy of the list of members of the Medical Council of India was sent to each member of the Working Committee of the I.M.A., as per its direction in connection with the question of non-recognition of the non-F. Sc. holders of the L.M.S. diploma of the Punjab State Medical Faculty by the Medical Council of India. This was noted.

4(18): *G. P. Notes and Government Securities of the I.M.A.*: The Hony. General Secretary informed the house that interest on G. P. notes in the name of the I.M.A. had been recently realised. Government securities and other relevant papers had also been kept in safe custody with the Central Bank of India Ltd., Delhi. These were noted.

Item No. 5(*a*): *FORMATION OF NEW BRANCHES.*

The Hony. General Secretary informed the house that the following new local branches were duly formed in accordance with Rule No. 3-C(*a*) of the Indian Medical Association:

	Name of Local Branch	*Provincial Branch*	*No. of members*	*Date of formation*
1.	Alipurduar	Bengal ...	14	1- 4-1951
2.	Kirnahar	Bengal ...	7	1-10-1951
3.	Tripura	Bengal ...	11	1-10-1951
4.	Forbesganj	Bihar ...	8	1- 4-1951
5.	Mangaldoi	Assam ...	6	1-10-1951
6.	Labac	Assam ...	9	1-10-1951
7.	Wadhwan Surendranagar	Gujarat & Saurashtra ...	10	1- 4-1951
8.	Baripada	Orissa ...	7	1-10-1950

The formation of these local branches were approved.

Item No. 5(*b*): *REVIVAL OF BRANCHES.*

The Hony. General Secretary informed the house that since the last meeting of the Committee, no defunct or suspended local branches have been revived.

Item No. 5(*c*): *SUSPENSION OF BRANCHES.*

The Hony. General Secretary informed the house that the respective provincial branches have recommended for suspension of the following local branches as per Rule No. 10-B(*a*) and the convention followed in the interpretation of this rule:—

1. Anakapalli under Andhra Provincial Branch; 2. Chandmari and 3. Gushkara under Bengal Provincial Branch; 4. Bidar under Hyderabad Provincial Branch; 5. Jeora and 6. Bhind under Madhya Bharat Provincial Branch; 7. Jazpur, 8. Kandrapara, 9. Karaput, and 10. Salipur under Orissa Provincial Branch; 11. Jagadhari, 12. Kalka and 13. Ropur under Punjab Provincial Branch; 14. Thana under Maharashtra & Karnatak Provincial Branch; 15. North Arcot under South India Provincial Branch; and 16. Ghaziabad under Uttar Pradesh State Branch.

The above-named local branches were declared to be suspended with effect from October 1950. The Committee also directed that if any of these branches were revived later on, the C.F.C. for these branches for the period under suspension would not be calculated as realisable dues.

Item No. 6: *CONSIDERATION OF C.F.C. ARREARS.*

The Hony. General Secretary stated that the following C.F.C. arrears of some local branches and other outstandings were unrealisable and, on the recommendation of the respective Provincial Branches and the Auditors, he suggested that these amounts be written off:

(*i*) Cuttack Local Branch (Under Orissa Provincial Branch)—Rs. 140/- (C.F.C. Arrears of 1950-51); (*ii*) Bellary Branch (Direct)—Rs. 160/- (C.F.C. Arrears of 1950-51); (*iii*) Other outstanding dues as recommended by the Auditors:—The Central Bank of India Ltd.; Rs. 354/3/6 Debit.

After some discussion, the following resolution was unanimously passed:—"Resolved that the above-mentioned unrealisable C.F.C. arrears and outstanding dues be written off subject to subsequent approval of the Central Council, as per Rule 13-C(*e*) (*v*) of the I.M.A."

Item No. 7: *ADOPTION OF (QUARTERLY) AUDITED ACCOUNTS.*

The following resolution was unanimously passed:—"Resolved that the audited accounts of the Central and Journal Departments of the I.M.A. for the quarter ending the 30th June, 1951, as circulated, be adopted."

Regarding the financial position of the Central and Journal Departments of the financial year ending on the 30th September, 1951, the Hony. General Secretary informed the house that accounts of the Central and Journal departments would show a surplus. The Committee thanked the authorities of Provincial and Local Branches, the Central Office and the Editor and the members of the Journal Committee for their co-operation and assistance in stabilising the financial position of the I.M.A. during the year 1950-51.

Item No. 8:—*SELECTION OF DELEGATES FOR THE INDIAN SCIENCE CONGRESS TO BE HELD AT CALCUTTA FROM 2ND TO 8TH JANUARY, 1952.*

The Hony. General Secretary read out a list of names recommended by the various provincial branches for this purpose.

On the suggestion of Dr. A. P. Mittra, the following members were unanimously elected as delegates of the Indian Medical Association:—

1. Dr. A. D. Mukharji (Calcutta).
2. ,, A. K. Sen (Calcutta).
3. ,, P. K. Guha (Calcutta).
4. ,, A. C. Ukil (Calcutta).
5. ,, P. K. Chatterjee (Calcutta).
6. ,, M. L. Biswas (Calcutta).
7. ,, A. N. Roy (Calcutta).
8. ,, K. K. Sengupta (Calcutta).
9. ,, Sukomal Sen (Calcutta).
10. ,, B. Narayana (Patna).
11. ,, G. K. Ghosh (Patna).
12. ,, M. A. Hai (Patna).
13. ,, Shyambehari Lal (Patna).

Item No. 9(*a*): *REPORT OF DR. S. C. SEN RE: THE 5TH GENERAL ASSEMBLY OF THE W.M.A. HELD AT STOCKHOLM, SWEDEN, FROM 15TH TO 20TH SEPTEMBER, 1951.*

Dr. S. C. Sen, the Hony. General Secretary and the representative of the I.M.A. to the W.M.A. gave a brief account of the proceedings of the last session of the General Assembly of the World Medical Association. This was noted.

Item No. 9(*b*): *REPORT OF DR. S. C. SEN ON OTHER ACTIVITIES DURING HIS RECENT TOUR ABROAD.*

Dr. Sen gave a brief account of his recent tours abroad, particularly in the Scandinavian countries. He was full of praise for the members of the medical profession and the medical and public health administration of the Scandinavian countries. This was noted and Dr. Sen's activities abroad were highly appreciated by the members present.

Item No. 10: *CLARIFICATION OF THE PROVIDENT FUND RULES OF THE ASSOCIATION'S EMPLOYEES PASSED BY THE WORKING COMMITTEE AT ITS MEETING HELD AT CALCUTTA ON THE 26TH AND 27TH FEBRUARY, 1944 [Resolution No. 15 (b)].*

The Hony. General Secretary stated that in view of the fact that the Provident Fund system could not be started for the employees of the Central Office earlier, on account of prolonged correspondence with the postal authorities and some other factors, it was, therefore, necessary for the Working Committee to direct whether the employees of the

Central Office would be entitled to the benefits of the contributory Provident Fund, with retrospective effect.

After a general discussion in which Dr. S. C. Sen, Dr. P. K. Guha, Dr. A. K. Sen and other members took part, the Committee unanimously resolved that the employees of the Central Office would be entitled to the benefits of the contributory Staff Provident Fund, with retrospective effect, from the date of confirmation of their appointments. It was further resolved that while preparing the budget for the year 1951-52, the Association's share of the contribution to the Staff Provident Fund for the period prior to the current year would have to be included in the expenditure under the appropriate head, to enable the Hony. General Secretary to incur the additional expenditure for this purpose.

Item No. 11: *CONSIDERATION OF A LETTER DATED 8-8-51 FROM DHANBAD BRANCH AND A LETTER DATED 9-8-51 FROM DR. B. V. MULAY RE: REFUSAL OF SOME INDIAN RAILWAY AUTHORITIES TO ACCEPT CERTIFICATES ISSUED BY PRIVATE MEDICAL PRACTITIONERS TO RAILWAY EMPLOYEES.*

Dr. B. V. Mulay and Dr. S. C. Sen explained the matter and it was noted. The Hony. General Secretary was authorised to take further action in the matter.

Item No. 12: *CONSIDERATION OF A LETTER DATED 17-8-51 OF DR. B. V. MULAY RE: A RESOLUTION PASSED BY THE HEALTH MINISTERS' CONFERENCE ON THE TRAINING OF VILLAGE VAIDS.*

The Hony. General Secretary explained the matter. A general discussion followed in which most of the members took part. Dr. B. V. Mulay was then requested to draft a course of training for a year or so for the auxiliary health personnel. Dr. Mulay agreed to take up the task.

Item No. 13: *CONSIDERATION OF A LETTER DATED 7-8-51 OF THE PUNJAB STATE BRANCH RE: A PROPAGANDA GRANT FROM THE INDIAN MEDICAL ASSOCIATION.*

Dr. A. P. Mittra, the Hony. Joint Secretary at Delhi, read out the relevant correspondence. Dr. Ram Rakha Dheer, the representative of the Punjab State Branch suggested that a sum of Rs. 500/- be sanctioned by the Committee for propaganda and organisation work by his State branch. Dr. S. C. Sen informed the house, in this connection, that the Punjab State Branch were likely to have a surplus of about Rs. 3,000/- at the end of the last financial year. Moreover, Dr. Sen added that the Journal Department had to spend a sum of Rs. 13/- approx. per member per year, though it was entitled, as per rules, to get Re. 1/- only, per member per year, as Journal quota of the C.F.C. which amounted to Rs. 8/- only per member per year. This showed the poor and unstable financial position of the Central Office. Dr. A. P. Mittra further informed the house that when the Provincial quota of the C.F.C. was raised from Rs. 2/- to 3/-, per member per year, it was expected that henceforth Provincial Branches would not expect sanction of any special grant from the Central funds of the Association, for propaganda purposes, unless it was absolutely necessary.

After a general discussion, Dr. R. R. Dheer and his Provincial Branch were requested to place before the Central Office, a detailed scheme on the subject, so that the matter could be discussed at a later meeting of the Working Committee.

Item No. 14: *CONSIDERATION OF A LETTER DATED 2-8-51 OF THE UTTAR PRADESH STATE BRANCH RE: THE INVITATION OF LUNCKNOW BRANCH TO HOLD THE ALL-INDIA MEDICAL CONFERENCE IN DECEMBER, 1953.*

Dr. P. K. Guha reminded the house, in this connection, that the Indian Medical Association would complete 25 years of its existence by December, 1953 and the Silver Jubilee of the Association would have to be celebrated during the All-India Medical Conference to be held in December 1953.

The Working Committee thanked the Uttar Pradesh State Branch and the Lucknow Branch for their kind invitation and decided that, in view of its importance, the matter should be finally decided at the Ahmedabad meetings of the Working Committee and Central Council, when all Provincial Branches were expected to be represented.

Item No. 15: *CONSIDERATION OF A LETTER DATED 3-8-51 OF SOUTH INDIAN PROVINCIAL BRANCH RE: AN INSURANCE SCHEME.*

The Hony. General Secretary explained the matter and it was noted.

Item No. 16: *ELECTION OF THE PRESIDENT AND 3 VICE-PRESIDENTS OF THE INDIAN MEDICAL ASSOCIATION FOR THE YEAR 1951-52.*

The President appointed the following members as scrutineers:—(*i*) Dr. S. Samaddar; (*ii*) Dr. Damodar Prasad; (*iii*) Dr. B. K. Vinchure and (*iv*) Dr. P. N. Sinha.

Before the ballot papers were opened, the President clarified the rules in this connection.

After the votes were counted by the scrutineers, the President announced the following results of the election:—

FOR THE OFFICE OF THE PRESIDENT:

S. No.	*Name of Candidate.*	*No. of votes.*	
1.	Dr. T. S. Tirumurti (Madras) ...	95	*Elected*
2.	,, A. C. Ukil	53	
3.	,, B. N. Ghosh	16	
4.	,, (Capt.) P. B. Mukerji ...	11	
5.	,, Major General D. P. Goil ...	9	
6.	,, A. C. Banerjee	6	
7.	,, (Capt.) R. C. Goulatia ...	5	
8.	,, J. C. Banerjee	2	
9.	,, B. L. Kapur	1	
	Invalidated	3	
	Not voted	1	
	TOTAL ...	202	

FOR THE OFFICE OF THE 3 VICE-PRESIDENTS:

S. No.	*Name of Candidate.*	*No. of votes.*	
1.	Dr. A. K. Sen (Calcutta) ...	68	
2.	,, M. D. Patel (Ahmedabad) ...	65	*Elected*
3.	,, Jawaharlal Rohatgi (Kanpur)	43	
4.	,, J. C. Banerjee	33	
5.	,, (Miss) M. P. John ...	29	
6.	,, R. B. Sathe	29	
7.	,, A. C. Ukil	24	
8.	,, M. Atal	22	
9.	,, D. V. Venkappa ...	22	
10.	,, (Capt.) P. B. Mukerji ...	20	
11.	,, P. Kutumbia	17	

12.	,, G. V. Joshi ...	...	16
13.	,, Tirumal Rao ...	...	15
14.	,, K. K. Sengupta	...	14
15.	,, B. R. Patel ...	...	12
16.	,, (Capt.) R. C. Goulatia	...	12
17.	,, Rajeswar Prosad	...	10
18.	,, Melkote ...	...	10
19.	,, P. S. Gupta ...	...	9
20.	,, R. Banerjee ...	...	9
21.	,, B. L. Kapur ...	...	9
22.	,, T. K. Dayalu ...	...	8
23.	,, P. Subramanian ...	...	8
24.	,, C. S. Thakkur ...	...	8
25.	,, C. P. Bhatt	...	8
26.	,, S. K. Ghosh Dastidar	...	7
27.	,, Ahmed Bux ...	...	7
28.	,, H. C. Barooah ...	...	7
29.	,, K. L. Pathak ...	...	7
30.	,, V. Nan ...	...	6
31.	,, B. B. Bhattacharya	...	6
32.	,, K. N. Misra ...	...	5
33.	,, Hanumantha Rao	...	5
34.	,, Faqir Chand Shori	...	4
35.	,, Chhabil Das ...	...	4
36.	,, J. L. Caroli ...	...	3
37.	,, P. Bhattacharjee	...	3
38.	,, Ram Rakha Dheer	...	2
39.	,, H. C. Dhupia ...	...	1
40.	,, D. C. Behal ...	...	1
41.	,, M. V. Ramana ...	...	1
42.	,, S. R. Mittra ...	...	1
43.	,, K. N. Lahiri ...	...	1
44.	,, R. K. Malhotra ...	...	1
45.	,, P. Satyanarayan	...	1
46.	,, S. D. Vohra ...	...	1
47.	,, Hiralal ...	...	0

Even though Dr. Chamanlal Mehta and Dr. A. D. Mukharji were not candidates, one vote was given to each of them.

The scrutineers informed the house that 4 ballot papers had to be invalidated in accordance with rules.

The President then thanked the scrutineers and declared that the following members were duly elected:—

PRESIDENT: Dr. T. S. Tirumurti of Madras.

VICE-PRESIDENTS: 1. Dr. A. K. Sen of Calcutta—(Senior Vice-President); 2. Dr. M. D. Patel of Ahmedabad, and 3. Dr. Jawaharlal Rohatgi of Kanpur.

Item No. 17: *CONSIDERATION OF A LETTER DATED 16-8-51 OF THE GUJARAT & SAURASHTRA PROVINCIAL BRANCH RE: FIXATION OF DATES OF THE WORKING COMMITTEE, CENTRAL COUNCIL AND ALL-INDIA MEDICAL CONFERENCE TO BE HELD AT AHMEDABAD IN FEBRUARY, 1952.*

Dr. A. P. Mittra, Hony. Joint Secretary read out the relevant letter.

The Hony. General Secretary who was authorised by the Committee to finally fix up the dates in consultation with the Gujarat & Saurashtra Provincial Branch, informed the house that the following dates had been provisionally fixed up:—

Working Committee—13th and 14th February, 1952; *Annual Meeting of the Central Council*—15th and 16th February, 1952; and *All-India Medical Conference*—17th, 18th and 19th February, 1952.

Dr. S. Samaddar requested the Hony. General Secretary to try to start the meetings on the 11th, so that the Conference could be finished by the 17th February. This was noted by the Hony. General Secretary.

Item No. 18: *CONSIDERATION OF THE QUESTION OF HOLDING A MEETING OF THE WORKING COMMITTEE IN DECEMBER, 1951.*

After a general discussion in which Dr. S. C. Sen, Dr. A. C. Ukil and others took part, it was unanimously decided that it was necessary for the Working Committee to hold its next meeting in December, 1951. Invitations to hold this meeting were extended on behalf of the Bengal and Bihar Provincial Branches at Calcutta and Jamshedpur respectively. After some discussion, the Committee unanimously fixed JAMSHEDPUR in Bihar as the venue of the said meeting. It was decided to hold the meeting on 24th and 25th December, 1951 at the time of the next Bihar Provincial Medical Conference also to be held at Jamshedpur.

Item No. 19: *CONSIDERATION OF THE RESOLUTION OF THE COIMBATORE BRANCH RE: DISAFFILIATION OF THE BRANCH FROM THE I.M.A. AND A LETTER OF DR. U. KRISHNA RAO ON THE SUBJECT.*

Dr. A. P. Mittra, the Hony. Joint Secretary read out the relevant correspondence.

The Working Committee, after a detailed discussion, regretted that the Coimbatore Branch still wanted to reduce the rate of C.F.C., though the matter was discussed in detail, from all possible aspects, at the Coimbatore meeting of the Working Committee held in October 1950. In view of the increasing cost of maintaining the Central and the Journal departments of the Association, the Committee expected that the Coimbatore and such other branches would realise that it was impossible for the Central Council to reduce the present rate of C.F.C. of Rs. 8/- only per member per year.

On a suggestion from Dr. U. Krishna Rao, the President of the South Indian Provincial Branch, the final decision of the matter was, however, postponed till the next meeting of the Working Committee when the representatives of the said Provincial Branch were expected to be present at the meeting.

Item No. 20: *CONSIDERATION OF A LATTER DATED 1-9-51 FROM DR. P. K. GUHA RE: THE PROCEDURE TO BE ADOPTED BY THE AD-HOC COMMITTEE IN FORMULATING THE NEW RULES OF THE INDIAN MEDICAL ASSOCIATION.*

Dr. P. K. Guha explained the matters concerned. He also informed the house that Capt. P. B. Mukerji, Dr. R. Sinha and some other members were invited by the Ad-hoc Committee to assist the Committee in its decisions. This was approved.

Dr. Guha further suggested that if the Association wanted to adopt the new set of rules at the next annual meeting of the Central Council, the Ad-hoc Committee should be requested to finalise their draft at an early date. The President agreed with the views expressed by Dr. Guha and suggested that the draft new rules should be prepared at an early date, so that copies of these could be made available to the mem-

bers of the Working Committee before the next meeting of the Committee to be held on the 24th December, 1951.

A general discussion followed in which Dr. S. C. Sen, Dr. B. K. Vinchure, De. A. K. Sen, Dr. J. Mojumdar, Dr. M. K. Roychaudhury and others took part. The house ultimately accepted the suggestion of the President.

Item No. 21: *CONSIDERATION OF THE CORRESPONDENCE BETWEEN THE SECRETARY, PUNJAB STATE BRANCH AND DR. A. M. DEVA, A JUNIOR VICE-PRESIDENT OF THE BRANCH RE: LICENTIATES, BENEVOLENT FUND AND THE REPORTED SPEECH OF THE PRESIDENT, I.M.A., AT HYDERABAD-DN AND ACCEPTANCE OF DR. DEVA'S RESIGNATION BY THE BRANCH.*

Dr. A. P. Mittra, Hony. Joint Secretary read out the relevant correspondence. Dr. Ram Rakha Dheer of Punjab further explained the matter. After some discussion, the action taken by the Punjab State Branch and the Central Office in the matter was approved by the house.

Item No. 22: *CONSIDERATION OF THE CORRESPONDENCE BETWEEN DR. CHAMANLAL MEHTA AND THE PRESIDENT, ROBERTSON MEDICAL SCHOOL, NAGPUR, REGARDING THE THREE YEARS' COURSE FOR MEDICAL AND HEALTH ASSISTANTS AND THE STRIKE BY THE STUDENTS OF THE SCHOOL.*

Dr. A. P. Mittra, Hony. Joint Secretary read out the relevant correspondence. The matter was further explained by Dr. Mehta and Dr. Vinchure.

The Working Committee regretted to note that the Government of Madhya Pradesh had recently decided to register, under the State Medical Registration Act 1916, the names of those who would complete the 3 years' course for medical and health assistants from the Robertson Medical School, Nagpur, after three years of satisfactory and approved service. The Committee recalled, in this connection, that the State Government concerned gave an assurance to the Indian Medical Association that persons trained at the said school, would never be accepted as registered medical practitioners, in the best interests of the people of the State. Dr. Mehta also pointed out that the Madhya Pradesh State Government agreed with the Indian Medical Association that the 3-years' training of the students of the Robertson Medical School could not possibly satisfy the minimum conditions necessary for the proper training of registered medical practitioners.

After a general discussion in which most of the members present took part, the following resolutions were unanimously passed:—

"This meeting of the Working Committee of the Indian Medical Association notes, with much regret, the recent decision of the Government of Madhya Pradesh in amending the schedule of the Provincial Medical Act of 1916 to bring the students passing out of the three years' Medical and Health Assistants' Course of the Robertson Medical School at Nagpur into the Provincial Medical Register as registered medical practitioners.

"The Association is further of considered opinion that such action of the State Government contravenes the assurance previously given by them to the Indian Medical Association and is a retrograde step which is likely to jeopardise the best interests of the people of Madhya Pradesh State in the sphere of medical relief."

Item No. 23: *CONSIDERATION OF "THE FIRST FIVE-YEAR PLAN," ESPECIALLY ITS CHAPTERS 15 TO 20 ON HEALTH, HOUSING, EDUCATION AND SCHOOL WELFARE ETC., PREPARED BY THE PLANNING COMMISSION OF THE GOVERNMENT OF INDIA.*

Dr. Chamanlal Mehta and Dr. S. C. Sen explained the matter and suggested that the matter being important be taken up and discussed threadbare at the next meeting of the Committee. The suggestion was accepted.

Item No. 24: *CONSIDERATION OF A LETTER DATED 1-6-51 OF THE SECRETARY-GENERAL OF THE WORLD MEDICAL ASSOCIATION RE: RECOMMENDATION TO THE W.H.O. NAMES OF PERSONNEL FOR APPOINTMENT IN EXPERT COMMITTEES SPONSORED BY IT.*

The Hony. General Secretary placed before the house the names suggested by the various provincial branches. After a general discussion, the Committee appointed a small sub-committee consisting of Dr. T. N. Banerjee, Dr. Chamanlal Mehta, Dr. A. C. Ukil and Dr. S. C. Sen for the final selection of names for the expert committees. The Hony. General Secretary was also authorised to send the names of selected persons to the World Medical Association, on behalf of the Indian Medical Association.

Item No. 25: *CONSIDERATION OF A LETTER DATED 8-9-51 OF THE SECRETARY, EAST GODAVARI BRANCH (UNDER ANDHRA PROVINCIAL BRANCH) RE: THE RESOLUTION OF THE WORKING COMMITTEE PASSED AT COIMBATORE MEETING IN OCTOBER, 1950 TO PUBLISH THE NAMES OF DEFAULTER MEMBERS IN THE JOURNAL OF THE INDIAN MEDICAL ASSOCIATION.*

Dr. A. P. Mittra, Hony. Joint Secretary read out the relevant correspondence. A general discussion followed in which Dr. A. K. Sen, Dr. A. C. Ukil, Dr. S. C. Sen, Dr. P. K. Guha, Dr. C. O. Karunakaran, Dr. M. K. Roychaudhury, Dr. S. N. Saxena, Dr. J. Mojumdar and others took part.

Dr. M. K. Roychaudhury then moved and Dr. J. Mojumdar seconded the following resolution:—"Resolved that the relevant resolution passed at the meeting of the Working Committee held at Coimbatore in October 1950 regarding the publication of the names of defaulter members in the Journal of the I.M.A. be rescinded."

Dr. P. K. Guha reminded the house that the Central Office could only arrange to publish the names of those defaulter members, list of which would be sent from the respective Provincial Branches for this specific purpose. Moreover, Dr. Guha suggested that the Central Office should refer such correspondence from Local Branches to the respective Provincial Branches for opinion, before these are discussed at the meetings of the Working Committee. The suggestions were accepted.

At the suggestion of Dr. A. K. Sen, the Senior Vice-President, the President ruled that the final decision of the matter be postponed till the next meeting of the Working Committee.

Item No. 26: *CONSIDERATION OF A LETTER DATED 18-9-51 FROM THE ASSAM PROVINCIAL BRANCH RE: FINANCIAL ASSISTANCE FROM THE I.M.A. FOR MEDICAL RELIEF WORK IN ASSAM.*

Dr. A. P. Mittra, the Hony. Joint Secretary, read out the correspondence and Dr. Bhubaneswar Baruah and Dr. Hem Baruah explained the matter. A general discussion followed in which Dr. S. C. Sen, Dr. S. Samaddar, Dr. A. C. Ukil and other members took part.

The following resolutions were then passed unanimously:

"Resolved that a sum of Rs. 2,500/- be donated from the funds of the Indian Medical Association to the Assam Medical Relief Committee of the Assam Provincial Branch of the Indian Medical Association, for medical relief work in the flood and earthquake affected areas of Assam.

"Resolved further that an amount of Rs. 1,075/- received from Uttar Pradesh and other donations received from Bihar and other Provincial Branches for this purpose, be also transferred to the account of the Assam Medical Relief Committee."

Item No. 27: *INTERPRETATION OF RULE No. 3-D(a) RE: THE REQUIRED MINIMUM STRENGTH OF MEMBERS TO MAINTAIN A LOCAL BRANCH.*

Dr. A P. Mittra, the Hony. Joint Secretary stated that when new local branches were formed, these had to have a minimum strength of five members, as per rules. But, at a later stage, in some branches, the membership strength came down to less than 5. The Central Office therefore, had to adopt an uniform procedure in interpretation of the relevant rule for application in such cases.

After a general discussion in which Dr. S. C. Sen, Dr. A. C. Ukil, Dr. A. K. Sen, Dr. P. K. Guha, Dr. A. D. Mukharji, Dr. C. O. Karunakaran and Dr. A. P. Mittra, took part, the President ruled that the interpretation of the Rule 3-D(a) would mean that a local branch with less than 5 members would be suspended at once and would cease to function from the next Association year or half year, unless the membership strength of the branch concerned was increased to 5 or more in the meantime. This was noted.

Item No. 28: *ANY OTHER BUSINESS WITH THE PERMISSION OF THE PRESIDENT.*

28(a): *Plantation Labour Bill*: Dr. A. P. Mittra, the Hony. Joint Secretary informed the Committee that at the request of the Cachar and Tinsukia Branches in Assam, he had made representations to Members of Parliament by personal interview, re: certain clauses in the Plantation Labour Bill which affected the medical personnel in the Tea Gardens. He also informed the Committee that Dr. S. P. Nath, Hony. Secretary, Cachar Branch, flew down to Delhi and personally contacted some Members of Parliament re: the matter on the eve of the Bill being taken up. The information given was noted.

28(b): *Extra Copies of J.I.M.A. supplement for Branches*: Dr. P. K. Guha suggested that extra copies of the *Supplement* of the J.I.M.A. be printed with effect from October 1951 issue, so that the Central office could circulate these to all branches for information and record. The suggestion was accepted.

28(c): *Back Numbers of J.I.M.A. for members*: Dr. P. K. Chatterjee suggested that arrangements should be made to despatch back copies of the J.I.M.A. to those new members, on whose behalf the respective Provincial Branches sent an intimation to the Journal office before the Association year or half-year commenced. The Hony. General Secretary informed the house, in this connection, that some of the Provincial Branches, particularly Uttar Pradesh and Bengal, have been following a practice of reservation of extra copies for their members. He added that it would be a debatable point whether these Provincial Branches would have any legal responsibility in paying off the C.F.C. of all these additional members, even if the expected number of extra members did not ultimately join the I.M.A. as members.

Dr. A. D. Mukharji and Dr. J. Mojumdar informed the house that, on an average, 500 extra copies were being printed for new members, by the Journal Department, at the commencement of the Association year. After some discussion the final decision of the matter was left to the Editor of the J.I.M.A. and the Journal Committee.

28(d): *Reduction of the rate of C.F.C. from Rs. 8/- to Rs. 6/- for displaced doctors*: Dr. Chamanlal M. Mehta said the Government had not yet sanctioned adequate loans to displaced doctors for their rehabilitation. He suggested that the Association should take up this matter with the Government and request the Government to sanction at least Rs. 5,000/- as loan to every displaced doctor. Dr. Mehta also suggested that the local branches should formulate a scheme of payment of subscription by the displaced doctors at a reduced rate. He suggested that the subscription should be reduced by 50 per cent, wherever possible, if the 50 per cent did not mean a rate lower than that of the C.F.C. These suggestions were accepted.

Dr. Mehta further suggested that the rate of C.F.C. should be reduced from Rs. 8/- to Rs. 6/- per member per year for those displaced members who would pay a lower rate of branch subscriptions for a period of three years. This would, of course, mean that the loss of Rs. 2/- would be equally shared by the Central and Provincial offices. This matter was referred to the Ad-hoc Rules Committee for opinion.

28(e): *Relaxation of Rules re. Import duty, Sales Tax and Excise duty on Essential Medicines and Appliances*: Dr. S. C. Sen suggested that the I.M.A. should approach the Government of India and the State Governments not to levy any import duty, sales tax and excise duty on certain essential imported medicines as antibiotics, vitamins etc., and appliances like X-ray plants and plates, instruments etc., not manufactured in adequate quantity in India. He informed the house, in this connection, that the Governments of Scandinavian countries had already adopted such a procedure in the interests of their people.

After a general discussion, the Hony. General Secretary was authorised to proceed in the matter and was requested to place his detailed scheme at the next meeting of the Committee, for final decision.

28(f): *Acknowledgments*: The following resolutions were proposed from the Chair and passed:—

(i) "The Working Committee of the Indian Medical Association places on record its deep appreciation and thanks for the hospitality and courtesy accorded to its members on the occasion of the meeting held at Gauhati on 14th and 15th October, 1951, by the Assam Provincial Branch and the Gauhati Branch of the Indian Medical Association."

(ii) "The Working Committee of the Indian Medical Association offers its grateful thanks to the authorities of the Bishop Cotton College for permission to hold the meeting of the Working Committee in their College premises at Gauhati."

With a vote of thanks to the chair, the meeting terminated.

Sd./- T. N. BANERJEE,
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Case 2. ADOLESCENT INSANITY-SCHIZOPHRENIA **Care of Dr..... MBBS.**

Female, Age 30. **Occupation :** Home work. **Complaints :** (i) Somebody will take away my ornaments to sell them away (ii) I wish that my brothers and father should be ruined (iii) Somebody is going to break my house etc. **Clinical Symptoms :** i) To sit silently and idle not to do any work (ii) no proper sleep (iii) a look of dum-idiot (iv) nothing abnormal was detected on physical exam-patient is a sterile-married life of 16 years. **Previous Treatments :** Nicotinic acid and injections and tablets in large doses for three weeks—no remarkable results. **Treatment with SILEDIN :** One tablet three times a day was given-with one bottle of fifty the mental condition was completely improved without leaving any trace of the former disorders—In all 100 tablets are given-patient has put up good amount of weight-looks very healthy and mentally-perfectly all right.

Case 3. ANXIETY NEUROSIS **Care of Dr..... LCPS. DPS. LM.**

Sex : Female, Age 42—**Occupation :** Household work. **Complaints :** Afraid to come out of bed-thinks she is very weak-at times irrelevant in talk-anxious look. **Clinical Symptoms :** Anxiety Neurosis. **Other Reports :** General debility-poor nourishment. **Treatment with SILEDIN :** 2 tabs. twice a day for one month. **Remarks :** After four days treatment the facial look of anxiousness improved-after a week complete improvement. Treatment was however continued for one month.

Case 4. SCHIZOPHRENIA **Care of Dr..... LCPS.**

Occupation : Male nurse in mental hospital. **Complaints :** Insomnia-fear as if Police is following-nearest and dearest appeared as enemies-suspicious as if he would be poisoned-blankness in head. **Clinical Symptoms :** Vacant face-dyspepsia-tender hepatic region. **Previous Treatments :** Electric shock Therapy-temporary improvements.

Treatment with SILEDIN : Has been taking **SILEDIN and LEPTADEN** for the last one month with definite improvement—no other treatment prescribed. **Remarks :** The patient says "there is great relief after taking medicine. I am taking regularly for the last 15 days. I am getting very good sleep. Indigestion is cured and get regular motions. Head remains very calm." After about two months he says : "I am feeling complete relief by the treatment."

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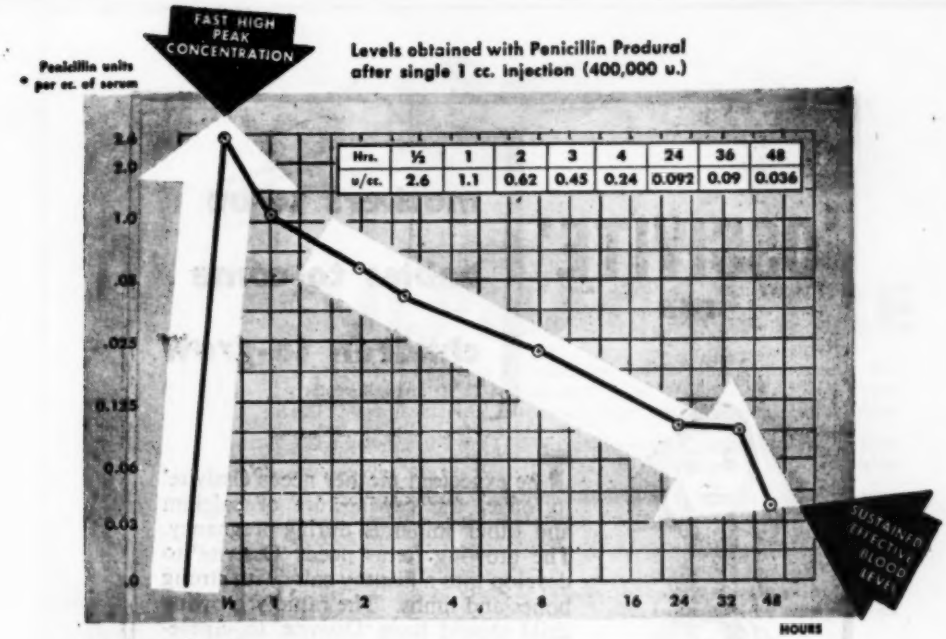

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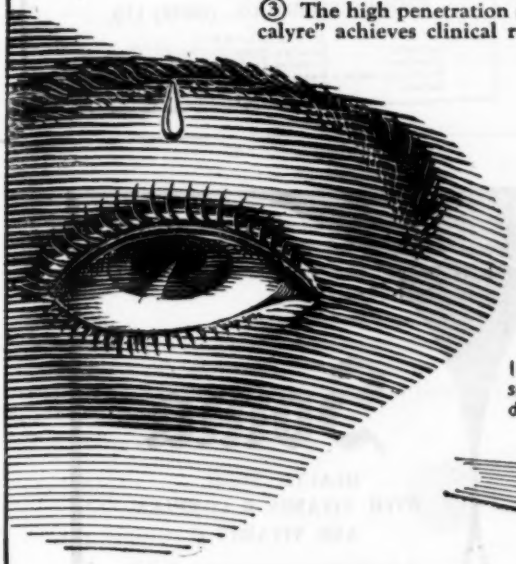

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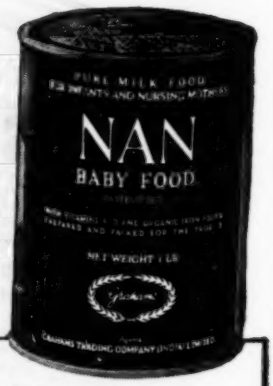

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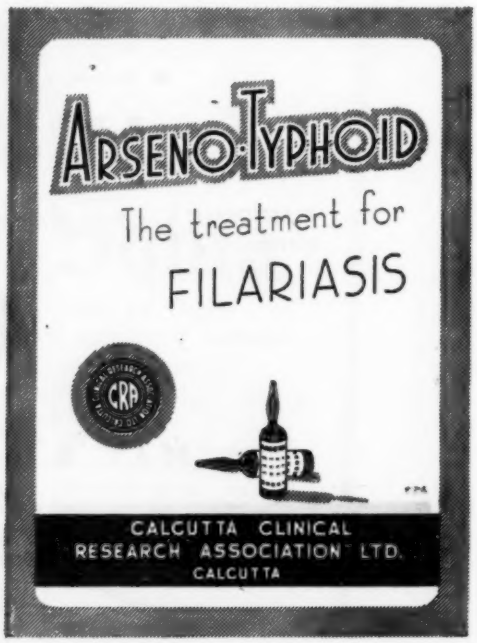

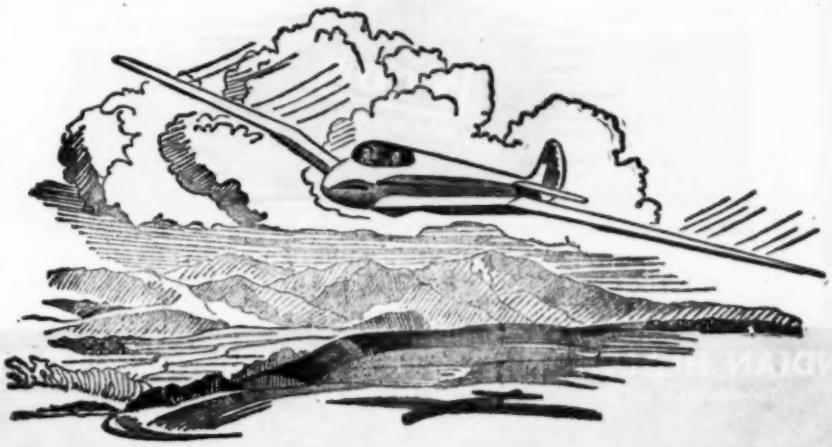

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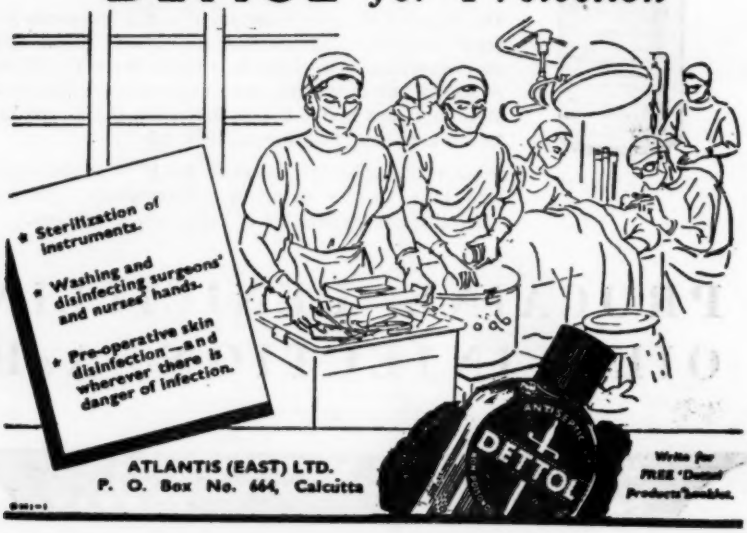
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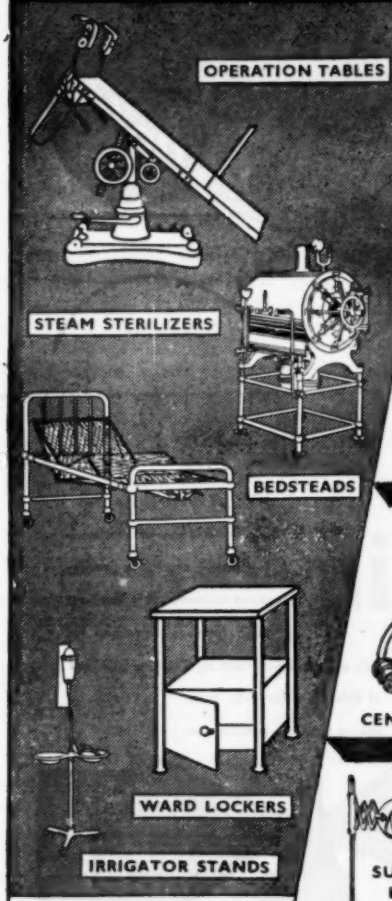

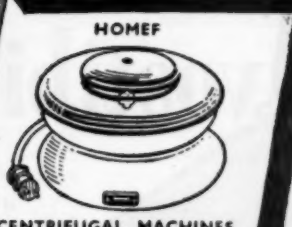

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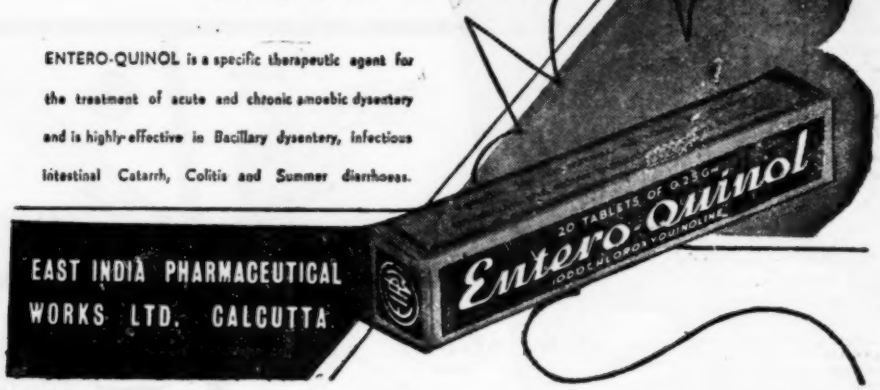

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Journal of the Indian Medical Association

VOL. XXI. No. 4 CALCUTTA JANUARY, 1952

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STUDIES ON CARBOHYDRATE METABOLISM IN TOXAEMIA OF PREGNANCY

1. SUGAR UTILISATION IN TOXAEMIA OF PREGNANCY

CHUNILAL MUKHERJEE,

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In 1912 Schirokauer first observed that alimentary glycosuria is a common feature of early pregnancy. Since then, considerable work has been done on the subject, and investigators have broadly divided themselves into two groups. (*i*) Those who believe that glycosuria is due to a lowering of the renal threshold (Nurnberger, 1921: Williams, 1923: Host, 1925: Faber, 1926: Gottschalk, 1927) Faber (1926, 1927) reported two normal primiparæ whose threshold fell from 150 and 200 mgm. per cent respectively to below 130 mgm. during the course of pregnancy. (*ii*) The second group of observers believe that the renal threshold may, or may not be lowered during pregnancy (Wallis, 1921: Labbe, and Chevki, 1926: Schmidt, 1928). In fact, Williams and Wills (1929) observed from a study of 640 glucose tolerence tests that the kidney threshold for glucose is not grossly lowered by pregnancy. This group of authors believe that the glycosuria of gestation is due to a defective utilisation of sugar in the body, which is probably of hypothalamico-pituitary origin. Evidence in support of the latter theory is found in the fact that pregnant women often show tendency to ketosis and ketonuria. Schultze (1926), and Bokelmann and Bock (1927) found that pregnancy is associated with an increase of blood lactic acid. Bokelmann ascribed this to a defective Cori cycle, and consequent delay in the resynthesis of lactic acid into glycogen. Histological (Hofbauer, 1907), and biopsy (Ingerslev and Teilum, 1945) studies of the liver during pregnancy have revealed centrilobular deposition of fat, which has been ascribed to glycogen deficiency.

Liver glycogen serves as an important reserve source of carbohydrates, readily available to all tissues of the body. In normal persons the supply is plenty, and a large amount can be stored in the liver in a few hours. In time of need this can be used with equal rapidity. The rate of this glycogenesis is dependent upon not only the glycogenic material available, but also the condition of the individual when this material is supplied. The rôle of the internal secretion of the pancreas is well-known in this connection. In order to observe the rate at which the sugar in the circulation is utilised by the body under favourable circumstances in toxæmic and normal pregnancy a series of experiments were undertaken. The manner of investigation and the results are presented below.

MATERIALS AND METHOD

The material for this study consists of 174 cases. These can be conveniently placed into four groups: All patients belonged to the same age group, 20 to 32 years.

Group 1. This consists of 26 non-pregnant women. These patients were of good general health, and admitted into the gynæcological wards for the investigation of dysmenorrhœa and sterility. None showed any evidence of organic or constitutional disease. They provided the values for the normal non-pregnant controls.

Group 2. This comprises of 48 cases of normal pregnancy. All were in the last trimester of gestation. This selection was considered necessary because the aim of the investigation was to study the results in late pregnancy toxæmias. Patients who showed any sign of organic disease, anæmia or toxæmia were excluded. These cases provided the control values for normal pregnancy.

Group 3. This includes 79 cases of hypertensive toxæmia of pregnancy, consisting of 23 cases essential hypertension complicating pregnancy, and 56 cases, where hypertension developed for the first time during the last trimester of gestation. Both types of cases had albuminuria and œdema. The second type has been described in the text as " true " pre-eclampsia.

Group 4. There are 21 cases of eclampsia in this group. In all patients the test was performed during the stage of convulsions and coma. No patient, where the last fit occured more than 2 hours before the test has been included for this study.

The test was performed as follows:

A sample of blood was obtained for the estimation of the basic blood sugar level. After disconnecting the

syringe, through the same needle was given an intravenous injection of 5 gm. of glucose (10 ml. of a 50 per cent solution), and 3 units of insulin. The small quantity of glucose used for this experiment was in order to avoid any loss due to glycosuria. The amount of insulin used was calculated to " cover " the amount of glucose injected. Blood from the opposite arm vein was collected 5 minutes, and 25 minutes after the injection. Blood sugar was estimated on these specimens.

In order to avoid glycolysis the blood was immediately transferred to isotonic sodium sulphate and copper sulphate solution (King, Pillai and Beall, 1941). Proteins were precipitated with Somogyi reagent (Somogyi, 1931) and the sugar was estimated in the filtrate by Folin's method. Photo-electric colorimeter was employed. Each estimation was done in duplicate, and where the results varied by more than 5 per cent, the estimation was repeated.

The bladder was emptied before, and at the conclusion of the experiment, and both samples of urine were examined for the presence of sugar. No patient, who had glycosuria, or developed glycosuria during the experiment, has been included in this series. Similarly, patients with evident hepatic, or renal disorder were excluded. All experiments were done under basal conditions, after a fast for 12 hours.

Results

Group 1—In 10 of the 26 non-pregnant females, a transient hyperglycæmia was caused by the injection (blood sugar above 140 mgm. per 100 ml.). However, normal blood sugar level was restored in all cases 25 minutes after the injection of glucose and insulin. The amount by which the blood sugar decreased during this period varied from 46·3 to 67·7 mgm., the average being 56·5±6·98 mgm. per 100 ml. In 84·7 per cent (22 out of 26 cases) of cases the amount of sugar utilised by the body was more than 50 mgm. per 100 ml.

Group 2—The peak level reached, as a result of the injection in pregnant women, was much more variable than that in the non-pregnant state. In 26 out of 46 cases of normal pregnancy the blood sugar after the injection reached the hyperglycæmic level (above 140 mgm. per 100 ml., Peters and Van Slyke, 1946). However, there was a fair correlation between the basic and ceiling levels of blood sugar in these cases. The hyperglycæmia was of a short duration, for in only 3 cases in this series did the blood sugar persist above 140 mgm. (143·5, 148·4, and 151·8 mgm. respectively) per 100 ml. 25 minutes after the injection of glucose and insulin. The fall in the blood sugar, which occured during this period varied from 22·9 to 58·3 mg. per 100 ml., with an average of 46·6±6·77. In 84·8 per cent (30 patients), more than 40 mgm. of blood sugar was removed from the circulation during the period of the experiment. In only 20·8 per cent of cases (10 patients) did this figure exceed 50 mgm. per 100 ml. In this respect the difference in the results between the non-pregnant and pregnant women is significant.

Group 3—In the group of 79 cases of hypertensive toxæmia of pregnancy the initial hyperglycæmia was observed in 45 patients. No relationship was found to exist between the height attained by the blood sugar as a result of the injection, and the severity of the toxæmia. The degree of hyperglycæmia in toxaemic pregnancy was not markedly different from that in normal gestation. The behaviour of the blood sugar during the subsequent period of the experiment showed considerable divergence in " true " pre-eclampsia and essential hypertension complicating pregnancy. These will therefore, be considered separately.

In 23 cases of essential hypertension complicating pregnancy the results were fairly allied to the values obtained in normal pregnancy. The range was almost identical. The maximum fall of blood sugar was 58·7 mgm., and the minimum, 21·2 mgm. per 100 ml. The average was somewhat less than that in normal pregnancy (41·2 mgm. ±7·16), but this is not of high statistical significance. The slightly lower average in essential hypertension is caused by a little shift to the left of the values obtained in this group of cases. In 87 per cent of patients these values were scattered between 30 and 50 mgm. Almost a corresponding scatter in normal pregnancy was found between 40 and 60 mgm. per 100 ml. Compared with normal pregnant women, 73·9 per cent (17 cases) showed less than average sugar utilisation.

The rate of removal of sugar from the circulation in pre-eclampsia is decidedly lower than that in either normal pregnancy or essential hyeprtension complicating pregnancy. In 92·3 per cent (52 cases) of this series of 56 pre-eclamptic subjects, the values obtained were actually less than the minimum observed in normal pregnancy. The minimum value was 2·8 mgm. per 100 ml., and the maximum 30·8 mgm. The latter was thus about 33 per cent less than the average for normal pregnancy. The average fall in the blood sugar in pre-eclampsia was 11·7 mgm. ±7·16, which is only about 25 per cent of the normal pregnant average. In only 4 cases (7·1 per cent) in this group the values approached the lower ranges of those in normal pregnancy, but even these were considerably less than the observed average for normal gestation. The difference in the sugar utilisation between normal pregnancy and pre-eclampsia shows high statistical significance.

Group 4—In 3 out of 21 cases studied here, the blood sugar exceeded 140 mgm. per 100 ml. immediately after the injection. During the subsequent period the rate of removal of the sugar from the circulation was extremely variable. In 11 cases it was less than 10 mg. and in 3 cases it was between 30 and 40 mgm. per 100 ml. In the remaining patients the values were scattered between these two groups. The average value obtained in eclampsia was 16·8 mgm. ±10·44.

An attempt was made to determine the cause of the high co-efficient of variation, which is 59·8. No definite relationship was observed between the number of convulsions and the rate of withdrawal of sugar from the circulation. In a patient with 14 fits the fall in the blood sugar was 32·6 mgm., yet in another patient, after only 2 fits the corresponding fall was 3·1 mgm. per 100 ml. The frequency distribution of the rate of sugar utilisation of all cases included in this study is presented in Table 1. The state of coma was found to be more closely related to the rate of sugar utilisation. Nine patients were in a state of deep coma when the experiment was performed. The decrease in blood sugar in these cases varied from 3·1 to 9·2 mgm. per 100 ml. A closer correlationship was observed between the carbon dioxide combining power of the plasma and the fall of blood sugar which occurred after insulin. This is shown in Table 2.

TABLE 1—SHOWING FREQUENCY DISTRIBUTION OF SUGAR UTILISATION IN NON-PREGNANT WOMEN, NORMAL PREGNANCY AND PREGNANCY TOXAEMIAS

Sugar utilisation	*Non-Preg.*	*Norm. Preg.*	*Pre-eclamp.*	*Essential hyper tens. Preg.*	*Eclampsia*
0 —10	...	...	25 (44·6%)	...	11 (52·4%)
10·1—20	...	...	27 (48·3%)	...	5 (23·8%)
20·1—30	...	1 (2·1%)	2 (3·6%)	2 (8·7%)	2 (9·5%)
30·1—40	...	8 (16·7%)	2 (3·6%)	12 (52·2%)	3 (14·3%)
40·1—50	4 (15·4%)	29 (60·4%)	...	8 (34·8%)	...
50·1—60	12 (46·2%)	10 (20·8%)	...	1 (4·3%)	...
60·1—70	10 (28·5%)	...	...	...	...
No. of cases ..	26	48	56	23	21
Average	56·8	46·3	11·7	40·2	16·8
Standard Deviation .	6·98	6·77	7·16	7·68	10·44

TABLE 2—FREQUENCY DISTRIBUTION OF SUGAR UTILISATION IN RELATION TO THE CARBON DIOXIDE COMBINING POWER OF THE PLASMA IN ECLAMPSIA. ESTIMATIONS WERE DONE WITH VAN SLYKE'S VOLUMETRIC APPARATUS.

Carbon dioxide combining power ml. CO_2%.	*Number of Cases Utilisation of sugar (mgm. per 100 ml.)* 0 —10	10·1—20	20·1—30	30·1—40
25 —30	1	...	...	...
30·1—35	9	3	...	...
35·1—40	1	1	...	...
40·1—45	...	1	1	...
45·1—50	...	...	1	3
Average	32·3	36·2	...	48·1
Min.—Max. ...	27·7—36·2	34·5—40·0	43·1—46·2	46·6—49·6

Sugar Utilisation and Concentration of Serum Gonadotrophins—The relation between the pituitary and the carbohydrate metabolism, and numerous observations indicating the presence of excess of gonadotrophins in toxæmia of pregnancy suggested that a study of the relationship between the concentration of gonadotrophins in the serum and the rate of removal of sugar from the circulation under the influence of insulin may be of interest. The manner of estimation of serum gonadotrophins, and a part of the study relating to their concentration in toxæmias of pregnancy have already been reported (Govan, Mukherjee, Hewitt and Harper, 1951).

The results of the present analysis are shown in Table 3. It will be seen that in toxæmia of pregnancy the extent of sugar utilisation decreases as the concentration of gonadotrophins in the serum increases. This is depicted not only in the average, but also in the range of values observed in each group. The difference in the values obtained show some degree of

statistical significance at all stages, but this becomes most marked when the titre of gonadotrophins in the serum rises above 1/2·5. In normal pregnancy the gonadotrophin concentration was always below this level, and up to this stage the rate of sugar utilisation showed no significant difference whether manifestations of toxæmia were present or not. A higher concentration of gonadotrophins was found only in the cases pregnancy toxæmia, and in this group sugar utilisation was markedly affected, becoming worse as the titre increased. The correlation between the two is not quantitative, but is extremely striking.

TABLE 3—SHOWING SUGAR UTILISATION IN RELATION TO THE CONCENTRATION OF GONADOTROPHINS IN THE SERUM IN NORMAL AND TOXAEMIC PREGNANCY.

	Titre of serum gonadotrophins					
	Neat	1/2·5	1/5	1/10	1/20	1/40
	Normal Pregnancy, 48 cases					
No. of cases	40	8	...	...	...	...
Sugar utilisation						
Average	46·8	43·8				
Min-Max.	34·5—58·3	22·9—54·0				
St. Dev.	6·21	7·13				
	Toxæmia of Pregnancy, 100 cases.					
No. of cases	12	18	12	27	25	6
Sugar utilisation						
Average	47·14	44·25	22·6	14·6	10·1	7·0
Min.-Max.	33·0—58·7	21·2—54·5	16·4—28·4	11·0—24·2	5·5—21·0	2·8—10·8
St. Dev.	5·83	7·98	4·46	4·94	4·11	3·77

DISCUSSION

The exact status of the carbohydrate reserve of the body in human pregnancy is not known. The only comparable work in animals in this direction is that of Schmidt and co-workers (1927). These authors estimated the glycogen content of the liver and muscles of "well fed" dogs during pregnancy. They found that gestation is accompanied with a fall of the glycogen content of the liver from 6 per cent in the non-pregnant state to 2·15 per cent. A similar reduction of the glycogen content of the muscles was also observed.

The obvious significance of these findings is the increased susceptibility of a pregnant animal to ketosis, a fact which applies as well to human beings. Toxæmia of pregnancy is attended with a greater risk of ketosis, and depletion of the alkali reserve of the body. Study of pregnancy toxæmias in animals show extensive fatty change in the liver with marked depletion of the liver glycogen. Fatal cases of human toxæmia of pregnancy also show fatty change and necrosis of the liver cells. The investigation undertaken here was aimed to demonstrate the speed with which sugar can be utilised by the body under favourable conditions. The findings show that the capacity for utilisation of sugar steadily decreases from the non-pregnant state to pregnancy, and deteriorates further as toxæmia of pregnancy develops. The difference between the non-pregnant and pregnant state is slight, but distinct. That, between normal and toxæmic pregnancy is more marked, in fact striking. It appears that toxæmia of pregnancy is not a homogeneous entity. The difference in the results obtained in this investigation in essential hypertension complicating pregnancy and pre-eclampsia is worthy of note. The utilisation of carbohydrates in the former condition is much more satisfactory than in the latter. This finding is in agreement with the differences observed between these two conditions with regard to serum gonadotrophin concentrations (Govan, Mukherjee, Hewitt and Harper, 1951), and the manner of response of the hypertension to methonium compounds (Mukherjee, 1950: Govan *et al*, 1951). Evidences are accumulating which show that the manner in which the physiological changes are affected in these two conditions are different.

Defective utilisation of sugar in toxæmia of pregnancy is an important feature of the disease. The part played by it in the occurence of œdema has already been suggested (Mukherjee and Govan, 1950). The recent experiences with cortisone in pregnancy toxæmia also throws light on the subject. (Moore *et al*, 1951).

However, the cause of this disturbed metabolism is not quite clearly understood yet. Factors which normally control utilisation and storage of sugars in the body are numerous, and among these the endocrines play an important part. Both anterior and posterior lobes of the pituitary body, the adrenal medulla and cortex, and to some extent œstrogens (Dolin *et al*, 1941: Ingle, 1948) have been known to have a contra-

insulin effect. Considerable controversy exists as to the functional status of the pituitary during pregnancy, and even less is known about this organ in toxæmia of pregnancy. Zondek (1931) believes that the pituitary lies dormant during pregnancy, while its function is taken over by the placenta. Severinghaus (1937, 1938) produced histological evidence demonstrating the presence of active secretory activity of the organ during the gestational state. Considering the changes in the adrenals and thyroid this seems probable. There are reasons to believe (Govan, 1951) that the pituitary is hyperactive in pre-eclampsia.

Smith and Smith (1939), and several other investigators have observed an increased concentration of gonadotrophins in the serum of patients suffering from toxæmia of pregnancy. Our own investigations described here, and in a previous communication (Govan *et al*, 1951) show that such a state exists in "true" pre-eclampsia, but need not necessarily be present in a patient with essential hypertension, who develop symptoms simulating pre-eclampsia. The relation between the concentration of serum gonadotrophins and the rate of sugar utilisation has been shown in the analysis presented above. An interpretation of these findings, and of the variations in the sugar utilisation in the non-pregnant state, and in various pregnancy conditions is possible in the light of the functional status of the pituitary. Cushing's (1912) earlier experiments, the Houssey animal (1931), and clinical investigations on pituitary basophilism (Dorfman *et al*, 1940: Albright, 1943) clearly indicate that pituitary overactivity can inhibit the effects of the pancreatic hormone. Thus, it seems probable that the disturbed carbohydrate metabolism, and decreased sugar utilisation observed in pre-eclampsia is related to a state of endocrine dysfunction, in which pituitary over-activity plays an important role either directly, or through the intermediary of the adrenal cortex.

Conclusions

The results of a series of experiments designed to study the rate of sugar utilisation in toxæmia of pregnancy are described. These are compared with similar results in non-pregnant women and in normal pregnancy.

The findings demonstrate that the rate of utilisation of sugar in normal pregnancy is slightly less than that in non-pregnant subjects. In toxæmia of pregnancy this is even less. Pre-eclampsia affects sugar utilisation in the body more adversely than essential hypertension complicating pregnancy.

Simultaneous study of the sugar utilisation and gonadotrophin concentration shows that the two are inversely related to each other. Available evidences suggest that the decreased sugar utilisation in toxæmia of pregnancy is due to a disturbance of the endocrine equilibrium, in which hyperfunction of the pituitary i an important feature.

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ALDEHYDE TEST IN PULMONARY TUBERCULOSIS

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INTRODUCTION

While going through the case histories of patients mostly from mofussil admitted in the hospital, a common story was encountered that their fever was thought to be due to kala-azar at first. The diagnosis, the patients explained, was based on enlarged spleen and a positive aldehyde test. For kala-azar the patients were given injections of antimony salts and with each injection there was a "reaction." By reaction it was meant that the temperature went up and there was an exacerbation of cough and sputum. It was seen that after some antimony injections, there was a spread of the lesion in lung. This only complicated the treatment of pulmonary tuberculosis for which the patient was admitted.

Enlarged spleen is not an uncommon finding in cases of pulmonary tuberculosis coming from the mofussil. The fever of pulmonary tuberculosis with the enlarged spleen not reacting to quinine may be mistaken for kala-azar. The present investigation was undertaken to find out the results obtained with the aldehyde test in cases of pulmonary tuberculosis.

MATERIALS FOR INVESTIGATION

100 cases were selected from the hospital for this test. Out of this 25 were early and 50 advanced cases of pulmonary tuberculosis, 20 were cases with clear fluid in chest and 5 of tuberculous empyema. This selection was made to investigate the aldehyde test results in almost all the common types of pulmonary tuberculosis. In each case the spleen and the liver were examined. Here enlarged spleen means spleens just palpable to spleens enlarged below the level of the umbilicus. The aldehyde test was done in each case according to the technique described by Napier (1943).

RESULTS

The results are given in the table. It will be seen that out of 100 cases, only 1 case was positive and this was a case of kala-azar complicated with pulmonary tuberculosis; and the diagnosis of kala-azar was confirmed by sternal puncture. 22 per cent had enlarged spleen and 17 per cent had enlarged liver. The test was negative in 91 per cent, doubtful (+) in 2 per cent and doubtful (±) in 6 per cent. The readings were taken according to Napier and compared with standards.

DISCUSSION

Napier (1943) in his interpretation of results of the aldehyde test was of opinion that positive and doubtful (+) results of aldehyde test with enlarged spleen of varying size should be interpreted as positive indication of kala-azar only when there are no obvious signs of advanced tuberculosis or leprosy. It was perhaps meant that in advanced tuberculosis and lepromatous leprosy the positive and doubtful (+) results may be obtained with the aldehyde test.

In the present series, aldehyde test was done in 50 cases of advanced pulmonary tuberculosis. Only in one case the test was positive and this was a case of kala-azar complicated with pulmonary tuberculosis. No positive results were observed in all other 49 cases. But 2 cases were doubtful (+), 4 cases were doubtful (±), 5 cases were negative (−) and 38 cases negative −ve.

TABLE

Condition of the lung	*No. of cases*	*Positive*			*Doubtful*		*Negative*		*No. of cases spleen enlarged*	*No. of cases liver enlarged*
		+++	++	+	(+)	(±)	(−)	−ve		
Early pulmonary tuberculosis	25	Nil	Nil	Nil	Nil	2	2	21	4	5
Advanced pulmonary tuberculosis	50	Nil	Nil	1	2	4	5	38	12	Nil
Pulmonary tuberculosis with clear fluid	20	Nil	Nil	Nil	Nil	Nil	3	17	5	2
Pulmonary tuberculosis with empyema	5	Nil	Nil	Nil	Nil	Nil	1	4	1	Nil
TOTAL ...	100	Nil	Nil	1	2	6	11	80	22	17

Positive: Solid, white, completely opaque (hard boiled egg). If complete opacity is produced in 20 minutes, the result is +++, if in 2 hrs. ++, if in 24 hrs., +

Doubtful (+): Solid, with milky appearance which looks opaque against a dark background, but shows the shape of window when held up to the light after 24 hrs.

Doubtful (±): Solid, slightly milky but quite transparent after 24 hrs.

Negative (−): Solid but crystal clear after 24 hrs.

Negative −*ve*. Serum unchanged (fluid) after 24 hrs. (Napier).

Even other cases of pulmonary tuberculosis did not give any positive results.

It seems safe to conclude that pulmonary tuberculosis alone, early or advanced, does not alter the chemistry of the blood in such a way as to give a fully positive aldehyde test. In cases where doubtful (+) or doubtful (±) results are obtained, treatment should be withheld till a confirmatory diagnosis of kala-azar is made after sternal puncture. In the mofussil it is desirable that practitioners should not diagnose a case as kala-azar if the result is doubtful (+) or doubtful (±) and when there is the slightest suspicion of lung tuberculosis. In any case, treatment with antimony salts should not be started in these cases even as a therapeutic test, as antimony injections flare up the lung lesions of tuberculosis.

SUMMARY

(1) The results of aldehyde test in 100 cases of pulmonary tuberculosis are presented.

(2) The test was positive in only one case of pulmonary tuberculosis with kala-azar, negative in 91 per cent, doubtful (+) in 2 per cent and doubtful (±) in 6 per cent.

(3) Pulmonary tuberculosis alone does not give a positive aldehyde test.

(4) In doubtful (+) and doubtful (±) cases, treatment with antimony should not even be started as a therapeutic test, if there is a suspicion of pulmonary tuberculosis.

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PRACTITIONERS' CORNER

SYMPOSIUM ON DIARRHOEA IN CHILDREN*

ETIOLOGY AND PATHOGENESIS

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DEFINITION AND SCOPE

While discussing the subject it is necessary to define the age group which one proposes to deal with. Our discussion will aim at covering the whole age period which begins with birth and is antecedent to adolescence, (age 0 to 14 years).

The term diarrhœa is easy to define. It indicates a symptom with a combination of two features, *viz.*, lessened consistency and increased frequency of the stools. Either of these can exist in the absence of the other; but a single character of the stool, lessened consistency or increased frequency, cannot constitute diarrhœa. It is known that in certain conditions increased frequency alone exists, *e.g.*, fragmentary constipation. Decreased consistency, likewise, may be observed by itself.

AETIOLOGY AND MECHANISM

In the causation of diarrhœa, diseases of the gastro-intestinal tract play as important part as the conditions arising outside this tract. Stimulation of the bowels leading to increased peristalsis is the important event in the production of diarrhœa. The stimulation may be of the small bowel or of the large bowel or of both. This may happen even in the absence of anatomical lesions in the intestine. With the stimulation of the bowel, contents may reach the colon, preventing absorption in the small intestine and/or causing greater pouring of fluid from the blood into the bowel.

Incidently I may mention that the greater stimulation of the small intestine tends to produce stools with acid reaction. These stools contain undigested food particles; mucus, if present, is intimately mixed with the contents and bile pigments present are in an unreduced state. The diarrhœa originating in the large intestine on the other hand, produces alkaline stools; the food is well digested; the mucus is free and the bile is reduced.

Diarrhœa occurs in a multitude of conditions; it is a symptom present in a large variety of diseased states. It is customary to attempt a classification of all these conditions; yet it must be admitted that a satisfactory classification is difficult to achieve. The bacteriological findings of the intestinal flora are not easy of interpretation; much less can these be correlated with any precision to the clinical manifestations to enable one to achieve a classification based on bacteriological studies only. Reviewing the morbid anatomy of the bowel in diarrhœa one will rarely find a situation more promising than the above. Leaving specific lesions as in amœbic and bacillary dysenteries, enteric fever, cholera

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etc., aside, the lesions in the bowel in the actute forms are phases of inflammation with a non-specific character.

The simplest classification would be from a clinical point of view, paying attention to the onset and the duration of the symptom. This will give us two large groups,—acute diarrhœa and chronic diarrhœa.

From the point of view of pathogenesis two ill-defined groups may be described *viz.*, simple diarrhœa and diarrhœa in intoxications and infections (toxic diarrhœa).

Taking the age of the patient as basis very useful groups can be made,—*e.g.*, diarrhœas of the new-born, diarrhœas of the infant, diarrhœas in children under two, diarrhœas under six weeks etc. These groups have immense practical value.

It must, however, be admitted that even using as our basis of classifications a combination of all the points of view stated above a comprehensive plan to serve all practical purposes is almost impossible.

Predisposing Causes

A number of conditions are presumed to be predisposing to diarrhœa.—

(*i*) Heat—it is a common experience that hot weather and warm climate often predispose to diarrhœa, whether associated with toxic conditions or not.

(*ii*) As regards the sexes diarrhœas occur with equal frequency in both the sexes.

(*iii*) Age plays a prominent part. Severe diarrhœas occur in the early years especially the first two years.

(*iv*) Individual susceptibilities have been noted as in the case of many other symptoms. Certain children are more susceptible and one attack weakens the resistance of the tissues of the bowel resulting in greater liability to recurring attacks.

(*v*) Social and economic conditions play a very big part. Diarrhœas occur more frequently in the children of the poor. Poverty carries with it numerous attributes, *e.g.*, overcrowding, bad ventilation, unclean homes, unsuitable foods, inadequate and unclean milk etc. It is needless to add that it is no wonder that diarrhœa occurs in almost 100 per cent of inmates of institutions and homes for children which are run with the minimum of funds and the minimum of care.

(*vi*) Kershaw (1947) has drawn attention to local chilling as one of the causative factors in acute non-specific diarrhœa and dysentery in infants.

This may serve as a non-specific determining cause.

Classification

I will now refer to the classification. Of the various classifications one may devise, the method of classifying diarrhœas into acute and chronic is all that one can reasonably undertake a discussion on.

Acute diarrhœa is a result of enteritis. The etiological factors are varied. Improper food, toxins, mineral salts, some kind of drugs, and bacteria are some of the prominent ones. Central or reflex stimulation of the bowel due to nervous influences may lead to frequency of stools, though there may not be pathological changes in the bowel wall.

Let us examine some of the causes of acute diarrhœa a little more closely. Indiscretion in diet, coarse food, improperly prepared food, insufficiently masticated food, food taken in too large a quantity, food taken too hot or too cold, food chemically or bacteriologically spoiled, are well-known causes of acute diarrhœa, almost at all ages except the very early period of life.

Acute diarrhœa may originate from ingested poisonous substances or drugs. Examples are arsenic, mercury, mineral salts, alkalies, acids, etc., and drugs like jalap and senna. All these produce direct irritation of the intestinal wall. They are capable of producing severe catarrh and even follicular necrosis. Acute diarrhœa can also be an accompaniment of several infectious diseases. Bacillary dysentery will rank as the foremost, cholera and typhoid can be separated as a group. But the diarrhœa as it accompanies infectious diseases like chicken pox, measles, smallpox, influenza and pneumonia form an interesting group for study.

In my opinion it is a result of ingested bacteria-laden contents of the oropharynx and upper respiratory tract coupled with lowered vitality.

Many of you who have been in practice from pre-antibiotic days have experience of the use of a little castor oil in babies having diarrhœa as a complication in acute infectious diseases.

A word may be said about the acute diarrhœa from nervous influences, the mechanism of which is far too complicated to be explained in a short compass. It may be added, however, that there is excessive stimulation of the nerves governing peristalsis and/or there is excessive pouring out of fluid in the intestinal tract thus involving motor and secretory factors.

Diarrhœa of the newborn and the infant. Speaking of acute diarrhœa and paying special attention to acute diarrhœa of early life, nutrition, climate and age, singly or jointly play the most important role. In acute diarrhœas of infant few causes beyond these can be described with confidence.

One group of diarrhœa in infants has been the source of headache to the clinician and the public health worker alike. This group is called by various names. Summar diarrhœa is the name more often used. It is a condition of gastro-enteritis of acute origin, is accompanied by vomiting and the whole affair is quite dramatic. Possibilities are that it is a virus disease.

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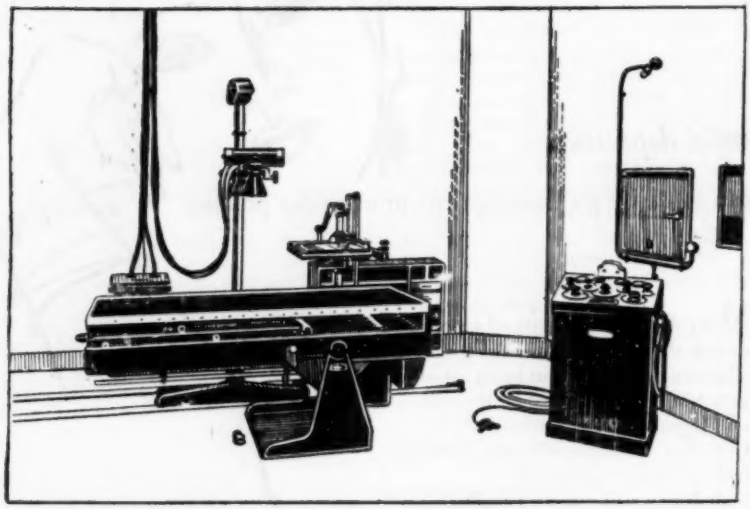

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The problem of acute diarrhœa in infants needs careful analysis. It is known that from 95 to 97 per cent of cases occur in bottle-fed infants.

I may give here a short account of the intestinal bacterial flora in the infant to have some idea of the relation of bacteria to the disease.

In the breast-fed this flora is relatively simple and consists largely of *Lactobacillus bifidus*. In the early weeks of life this bacillus constitutes 99 per cent of the total organisms in the fæces (Cruickshank, 1915). In addition there are a few enterococci and gram-negative coliform bacilli (Tissier and Dreyfuss, 1925).

The intestinal flora of the bottle-fed infants is not so simple. *L. bifidus* is uncommon. Another member of the same group *L. acidophilus* is present in large numbers (Tissier, 1900). Coliform bacilli, enterococci, gram-positive aerobic spore-bearing bacilli and anaerobic bacilli occur abundantly.

The bacterial flora of the grown child are more akin to that of the adult. The subject of the intestinal flora of the adult is a complex one and is irrelevant here.

I have said so much about the bacterial flora of the intestine for the simple reason that there has been a uniform failure in determining the bacterial origin of infantile diarrhœa (summar diarrhœa of infants). It may also be mentioned that some claim that the diarrhœa syndrome can be explained on the basis of invasion of the body by certain specific bacteria from the intestinal group.

The subject of diarrhœa in the new-born has received a great deal of attention. A number of epidemiological, clinical, bacteriological and experimental studies have been carefully carried out. Here I will briefly narrate some.

Pappenheimer and Cheever (1948) have studied the diarrhœal diseases in suckling mice. In the histopathological studies they have described cytoplasmic inclusion bodies in the intestinal epithelium in relation to the disease. This is a very interesting observation especially in the light of the following work in relation to human material.

Light and Hodes (1949) have carried out interesting studies on epidemic diarrhœa amongst infants under six weeks. In their work they have been unable to isolate any diarrhœa-producing known pathogenic bacteria from stools or the nasopharygeal washings. Inoculation of the stool or the nasopharyngeal washings intranasally, intraperitoneally and intracerebrally in mice, hamsters, guinea-pigs, rabbits, ferrets, cotton rats, and four-days-old kittens produced negative results. However, inoculation experiments in calves gave interesting positive findings. 20 c.c. of a bacteria-free filtrate (Seitz filtrate) were given intranasally to three-weeks-old quarantined calves. Two days after the inoculation mucoid diarrhœa commenced, in a few days it became bloody, on the 11th day the calf was almost prostrate, and died on the 15th day in fatal cases.

Experimental studies of inoculation on 89 calves have been published by these authors. There have been 13 per cent deaths in the experimental animals. In the non-fatal animals the duration of the diarrhœa has been 17 to 21 days. The calves were left debilitated.

The convalescent calves were immune to inoculation or to reinfection. Serum of these calves was capable of nutralising the homologous infective agent. It was likewise observed by these workers that neutralising antibodies were found in the serum of convalescent babies.

Autopsy findings in the fatal calves were as follows: œdema, hyperæmia, and hæmorrhage in the intestinal walls in all, mucus in the small and large bowel, definite ulceration of the large intestine in few, slight bronchopneumonia in one, a few subacute abscesses in the kidney in one. Inclusion bodies in the epithelial cells were not demonstrated. The autopsy findings are those of dehydration of 5 to 7 days' standing.

The infective agent has been studied by these workers in greater detail. It is partially resistant to heat. It is wholly inactivated by boiling for five minutes. It resists heat to 70°C for 60 minutes. Prolonged drawing or heating at 80°C for one hour attenuates the infective agent. Inoculation in calves of the attenuated agent produces a modified and mild form of the disease, *viz.*, mild diarrhœa after prolonged incubation period. Such a modified disease is followed by effective immunity.

Can one say that this work has sown the seed of a prophylactic inoculation?

I have detailed this work at some length on account of its importance. Taylor, Powell and Wright (1949) have studied clinical materials from six different epidemic outbreaks of diarrhœas in the new-born. They have also been unable to detect any known intestinal pathogenic bacterium. Their virus studies have been negative. They discovered a strain of *B. coli* D 433 in some.

Combined studies by several learned bodies investigated 28 outbreaks in New York (1949), 618 cases with 62 deaths were encountered between 1942-1947. The general conclusion of this study was that the condition was preventable to a certain extent by adopting greater care, more cleanliness, preventing visitors, etc.

Stern (1947) has discussed the prevention of epidemic neonatal diarrhœa.

Wright and Wright (1946) give an account of diarrhœa and enteritis amongst infants in the London area in the period 1930-1938.

Mukharjee (1947) has published observations on acute diarrhœa amongst infants during summer months in Jamshedpur and suburbs.

The most outstanding work appears to be in relation to virus origin. Pappenheimer and Cheever (1948) have produced histological evidence of the virus origin of epidemic diarrhœa in suckling mice. May be, there is a human parallel to this.

These are some recent pieces of work I have selected for placing before you.

Such detailed account of diarrhœa in the newborn and of the infant does not however exhaust our subject.

Besides these studies some sporadic bacteriological and other studies appear to have been made. They have all failed to produce any striking or helpful data. The only promising study opening the field for work is in relation to the virus.

Biochemical studies of the blood, stools, urine and other fluids of the body have an unquestionable status in planning treatment and assessing the clinical picture. I am leaving these to other speakers. I am sure they will have occasion to refer to these topics.

The problem of diarrhœas in children of the higher age groups. The pathogenesis of these approaches that of the adult.

I enumerate below the groups of causes of diarrhœa:

(*i*) Gastrogenous due to disturbed gastric function.
(*ii*) Due to disturbed pancreatic function.
(*iii*) Due to disturbed liver function.
(*iv*) Due to disturbed endocrine function as that of pancreas, suprarenal, thyroid etc.
(*v*) Due to cardiac and renal conditions.
(*vi*) Due to nervous influences, such as anxiety, excitement, organic nervous disease and the like.
(*vii*) Due to deficiency diseases.
(*viii*) Due to disturbances of metabolism, producing endogenous toxins.
(*ix*) Specific gastro-enteritis or enteritis,—viruses amœbic, bacillary, parasitic, helminthic, tuberculous etc., irritant drugs, chemicals etc.
(*x*) Conditions arising out of acute infectious diseases.
(*xi*) Non-specific enterocolitis and
(*xii*) Some unexplained gastro-enteritis.

I may add that this list of causative factors is neither in the order of frequency nor in the order of importance. At best it can aim at completeness.

Chronic Diarrhoeas

Having so far dealt with the subject of acute diarrhœa in early life let me, even more briefly, refer to chronic diarrhœas in early life.

Diarrhœa is a symptom which may become chronic. The incidence of chronic conditions rises as one goes up the age scale. Etiological factors are about the same as those in the acute forms probably modified. The bacterial aspect, the parasitic and helminthic content and the question of nutrition of the child attain greater importance in the consideration of chronic diarrhœas.

It would be more appropriate to speak of the diarrhœal syndrome in relation to the acute and the chronic forms alike. It would be unscientific to focus attention on the gastro-enteritis and neglect the child.

Acute diarrhœa of children may be of bacillary, helminthic or virus origin, it is the total picture of the condition that matters.

The picture I produced before you earlier in this paper of experimental work indicates that deaths of experimental animals have been due to dehydration. I have on my record a total of about 300 autopsies in the age group 0 to 10. A fair proportion of these have been on cases of enteritis or gastro-enteritis in infants about one year. The autopsy picture in most has been that of advanced dehydration showing the attendant morbid anatomy.

Some problems connected with diarrhœas in early life are typically public health questions. In countries with more money and with better public health organization, neonatal diarrhœa and diarrhœa amongst infants have been controlled with a distinct lowering of the mortality. Let me repeat, some of these diarrhœas if not all, are preventable.

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CLINICAL MANIFESTATIONS

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The clinical manifestations are varied. Its severity varies with age, season, and nutrition of the child. The younger age group presents higher incidence, both for the acute and chronic diarrhœas. In our experience, the incidence in the first year of life is almost four times that amongst the children five years old. The disease is almost equally distributed in both the sexes. Monsoon months provide the maximum number of acute diarrhœas. These may assume an epidemic form at a rapid and alarming rate, affecting children of all the age groups.

The clinical manifestations may be considered in three groups:

(*a*) Important symptoms of diarrhœas.
(*b*) Effects of diarrhœa.
(*c*) Clinical varieties.

Some of the Important Symptoms

The definition of diarrhœa is frequency of loos. stools. For its adequate appraisal, the normal type of stools in infants may be considered first. In a newborn baby, meconium consists of partially dried intestinal secretions and debris which have accumulated in the large intestines. For the first three or four days the baby passes three or four meconium stools daily. These are usually dark brown with greenish tinge, and of sticky consistency. In breast-fed babies, these change gradually, during the next few days, to golden yellow colour, having a slight aromatic odour. They are soft and are of semisolid consistency with acid reaction. The number of stools vary on an average, from 2-4 per day. In a healthy bottle-fed baby, 1 to 2 stools are passed in a day. These are better formed and contain more solid material. The colour is usually pale yellow. They should be perfectly smooth with a homogenous character and of neutral or alkaline reaction. The odour is slightly foul.

In diarrhœa the number of stools depends on various factors; thus quantitative or qualitative defects of diet, presence or absence of infection in the intestinal tract, tonicity of the intestinal musculature, and infection outside the alimentary tract may influence the frequency of stools. The more frequent the stools, the more liquid they are.

The diarrhœal stool is the result of an increased peristaltic action of the intestine. The normal amount of water is not absorbed, and sufficient time is not allowed for the formation of insoluble soaps which together with dead bacteria constitute the bulk of the solid portion of the stools; consequently, they are thin and liquid. The colour of stools in diarrhœa is usually green or yellow. In diarrhœa the green bile passes through the intestine so rapidly that it remains unchanged. Yellow stool if exposed to air for some time, often turns green on the surface due to a process of oxidation. This change of colour is of no significance. In order to assess the significance of the colour of the stool they must be examined fresh.

The number of stools will depend on the ætiological factors. In diarrhœa resulting from dietetic upsets and parenteral infection, the number of stools may not exceed six per day, while diarrhœa resulting from gastro-intestinal infection, may give rise to greater frequency of stools. In some children the frequency becomes so marked that the child is said to be almost continuously passing stools. The total volume of fluid lost may be great and out of proportion to the food and fluid intake. The inspection of diaper alone will not reveal the true quantity of fluid lost.

The character and colour of stools will depend on the precipitating factors. In starvation diarrhœa, the stools are numerous, greenish, small in quantity and contain little fæcal matter. They are passed every time the child is fed. In fact, this is a form of lienteric diarrhœa. Diarrhœa may also result from prolonged starvation therapy. In such cases frequency of stools will not yield to usual therapy, but institution of large feeds will result in prompt improvement. Infants who are overfed may have four to seven large, yellow and relaxed stools per day. Although they are loose they are fairly well digested, and have a foul odour.

Fatty stools result when the baby is receiving too much fat in the food. The stools are usually slimy, offensive, and greyish white in colour. Sometimes they are bulky and contain curd. The latter is due to soaps resulting from combination of fatty acids and calcium. If there is also too much of sugar, the stools are usually loose, frothy and contain many soft curds. Excess of sugar is the element in the infant's food that tends to make the stools loose. Infants who are unable to tolerate sugar, pass frequent, greenish, frothy, and sour, fermentative stools with considerable gas.

Both in fatty and carbohydrate dyspepsia, stools contain acids such as acetic and butyric. These acids produce excoriations of the buttocks, perineal regions, scrotum and prepuce. These regions become sore and red and defæcation is associated with considerable distress and crying. Involvement of prepuce will lead to dysuria. Constant rubbing of the buttocks with the napkins will tend to augment the soreness. In some babies the heels may also develop redness and soreness from constant contact.

The quantity of blood in diarrhœal stool varies greatly. While large amounts of blood present little or no difficulty, minute quantities will only be made out on microscopic or chemical examination. The presence of blood, mucus and pus in the stools usually indicates bacillary dysentery.

Mucus in small amounts may be seen normally in the stools, especially in breast-fed infants. A large amount of mucus both in breast and bottle-fed infants indicates irritation in the intestinal tract. Large lumps of mucus with or without blood may also indicate intestinal allergy. Mucus mixed with fæcal matter may be due to amœbic dysentery. It should be remembered that administration of barleywater and arrowroot kanji may also give slimy stools resembling mucus.

The manner of expulsion of stools will be of some guidance as well. Mild infection tends to lead to effortless evacuation. Intensely increased peristalsis, resulting from severe intestinal infection will force the stool out in a jet. Noisy, explosive stools are met with in dietetic upsets and in epidemic diarrhœa of the new-born.

Straining and/or tenesmus may accompany the passage of stools. This is particularly present in bacillary dysentery infection. Its presence indicates involvement of the rectum, and results in response to foreign body sensation. The process of straining will predispose to the abnormal descent of the anal canal with the resultant prolapse of the anus and rectum. In severe cases the anus becomes patulous.

Vomiting. Vomiting frequently accompanies diarrhœa. In the initial phase of diarrhœa the child may have only nausea or retching. But in moderately severe or toxic cases, it will form almost a constant feature. In the early stage of the diarrhœa, in some patients, it is so persistent, that oral nourishment or therapy will be impracticable. In some children, vomiting occurs for the first two or three days in the diarrhœal disorder while in others it may again become marked in presence of dehydration and acidosis at a later stage. In diarrhœa of dietetic upsets, vomiting occurs in the form of regurgitation. High fat content of diet delays gastric emptying and this predisposes to the production of irritant fatty acids, which induces vomiting. When vomiting is in excess, frequency of stools tends to be less. Vomiting may contain streaks of blood or it may even be of coffee-ground colour in which case the prognosis becomes grave.

Abdominal distension: Abdominal distension more frequently results from swallowed air than gases resulting from fermentation. The latter is largely due to accummulation of carbon dioxide, which is more quickly absorbed than air. Underfeeding leads to excessive swallowing of air which may produce distention of the stomach. In mild diarrhœa the abdominal contour may not show any departure from the normal. Distention of abdomen however, may be a marked feature in the severe type. This usually results from accumulation of gas in the intestine and general hypotonia. Extreme distension of abdomen will lead to upward pressure on diaphragm producing respiratory distress. With the moderate distension of the abdomen the coils of intestine will be seen standing out prominently. A doughy abdominal feel will suggest either tuberculous abdomen or infestation with worms. Severe distention may lead to paralytic ileus. In rare cases, dysenteric ulceration may perforate leading to localised or generalised peritonitis.

Pain. The history of pain is vague and unsatisfactory. In some cases it is complained of frequently, with no definite localisation, while in others it is in para-umbilical and descending colon regions. This type of pain tends to be griping in nature with frequent evacuation of the bowel.

Temperature. Rise of temperature may or may not be associated with the diarrhœal disorder. The temperature elevation will be slight or absent in mild type of diarrhœa, while a considerable rise should always arouse the suspicion of bacillary dysenteric infection or parenteral infection. In acute fulminating variety, the temperature may reach a dangerous level. However, it must be noted that the appraisal of the true rise in temperature may not be possible by the external examination alone, and in some cases, rectal examination will indicate a hot feel, and register high rise, in sharp contrast to the axillary temperature. In under-nourised and premature babies temperature elevation may be insignificant or absent.

Parenteral infection. It is well-known that foci of infection outside the gastrointestinal tract may give rise to frequency of stools in addition to other constitutional disturbances. Hence it is imperative that every child afflicted with diarrhœa should be subjected to a complete clinical examination at the earliest moment and these storm centres of infections should be located and adequate steps should be taken for their eradication. It is common experience that certain centres of infective foci will particularly predispose to diarrhœa. Thus rhino-pharyngitis, acute otitis media, bronchitis and bronchopneumonia, pyelitis, and skin lesions such as eczema and infected scabies are associated with diarrhœal disorders.

Effects of Diarrhoea

Diarrhœa may produce serious results, if it is of severe nature, or, prolonged in duration. The chief effects are (1) loss of weight resulting from starvation (2) loss of water causing dehydration (3) loss of mineral salts and fixed bases leading to acidosis or alkalosis (4) toxæmia resulting from bacteria and their products.

Loss of weight results from loss of appetite and inadequate intake of diet. Such food, as is taken, is only partially digested and absorbed. In such circumstances body tissues themselves are utilised for providing energy. The dehydration still further leads to loss of weight which may be as much as half to one pound a day.

Dehydration.—Vomiting and diarrhœa lead to rapid loss of fluid. Poor absorption of water will augment the state of dehydration. A relatively small

proportion of water of the body is within the cells of body tissues, and a large proportion is outside and around the cells. This interstitial water is labile and changes in amount with different degrees of hydration of the body. Infants dehydrate readily because they have approximately twice as much interstitial water in proportion to the body weight. The effects of dehydration are proportionate in severity to the degree of dehydration. In the early stage of dehydration the child will be restless and excited. The fontanelle will be slightly depressed, the tongue slightly furred and dry and the skin will exhibit loss of turgor. Severe dehydration will lead to extreme and rapid loss of weight. Coincident with the loss of fluid and weight the appearence of the patient changes rapidly. He looks acutely ill with lustreless and sunken eyes. The gaze is fixed in a stare, or, the eyes may be rolled up behind the half closed lids. The fontanelle is depressed; over-riding of the skull bones may be found in infants below six months. However it must be remembered that over-riding of the skull bones on each other will lead to narrowing of the fontanelle with masking of the depression.

Thirst is a constant feature. The lips are dry and mouth is often held open. The tongue becomes dry and furred. Stomatitis may result.

The skin is dry and loses its elasticity. It may be picked up in folds which flatten out slowly. Petechiæ and œdema when present indicate grave prognosis. The urine becomes scanty and concentrated, and it may contain small amount of albumin, sugar and casts. The output may cease completely and anuria may result. This may lead to symptoms of extrarenal uræmia exhibited by nausea, vomiting, irritability, restlessness, muscular tenderness and convulsions. Loss of fluids, hæmoconcentration and diminished renal cutput lead to increase in non-protein nitrogen of the blood. The dehydration itself may induce hyperpyrexia. Because of lowered blood volume the circulation is impaired, the pulse is small and rapid and irregular. The limbs become cold and cyanotic. With moderate dehydration the child is restless and excited. With greater degrees, however, prostration become marked and convulsion and coma may occur.

Acidosis is present in severe cases. It results from the failure to excrete acid substances such as acid phosphates which are normally excreted in the urine. Incomplete oxidation of food results in accumulation of acids, and this together with loss of base in the stool contribute towards occurrence of acidosis. With the onset of acidosis the breathing is greatly altered from the normal. Instead of being chiefly of the abdominal type, it becomes mainly thoracic with deep and rapid rate.

Toxæmia, resulting from bacterial toxins, will give rise to marked prostration, and some children die even before developing actual diarrhœa. In general, tachycardia with feeble and thready pulse result, and the child appears to sink in stupor. In some patients, however, bacterial exotoxins may produce signs of meningeal irritation and convulsion.

Clinical Types

Clinically the diarrhœal syndrome may be divided in (1) mild, (2) moderately severe, (3) severe, (4) acute fulminating, and (5) atypical varieties.

Mild. Diarrhœa resulting from temporary overfeeding, and parenteral infection are usually mild in character with short duration and little constitutional disturbances. Vomiting will frequently accompany overfeeding, while it will precede the onset of diarrhœa in patients with parenteral infections. The temperature elevation will be slight or absent; dehydration, acidosis and metabolic disturbances are absent. The stools are increased in number only moderately. Their consistency becomes soft or liquid. The colour usually varies from yellowish-green to green. These infants are usually fretful, irritable, and appear to suffer from abdominal pain. But the toxic manifestations are absent and the stools do not contain blood and pus. Response to therapy is uniformly satisfactory.

Moderately severe. This group includes those patients who display frequency of stools and vomiting with only the early manifestations of dehydration. Moderately high fever is almost of constant occurrence. Vomiting of only mild degree will be complained of, it being present only at the onset. Stools may number six or more per day. They are of liquid consistency and green in colour. The loss of fluid is just sufficient to produce loss of turgor of the skin, slightly depressed fontanelle, dry tongue and only slight diminution in output of urine. Metabolic disturbances and acidosis are usually absent. Constitutional disturbances in the form of irritability, fretfulness, abdominal colics and distentions are usually present. Convulsions, coma and circulatory collapse are not encountered in this variety. The patient responds very satisfactorily if the treatment is commenced early.

Severe Variety. The undernourished are especially prone to develop this variety. The incidence of parenteral infection tends to be high in these children. Not infrequently there is vomiting at the onset and it may become a troublesome feature. Fever of moderate or severe degree is almost constant. The stools may exceed ten per day and they tend to be more liquid, offensive and irritating. The total volume of stool may be very great and out of all proportion to the fluid and food intake. Small amount of blood is occasionally present in the stool but one should always think of bacillary dysentery infection when it is great in amount. Pus cells will be found in a fairly large number on microscopical examination, though naked-eye inspection may not reveal their presence. The evacuation of bowels may be preceded by pain, and tenesmus usually accompanies the act of defæcation.

Thirst is a common feature in all these patients, and dehydration is commonly found; its severity depending on the frequency and intensity of stools and vomiting. This may assume serious proportion leading the child to circulatory failure and collapse. Hæmoconcentration is invariably present and the renal output may diminish dangerously. Toxæmia and prostration become marked. In all these patients rapid and feeble pulse will be found. Acidosis and dehydration will tend to increase irritability, restlessness and vomiting. Anorexia is a contant feature and it often leads to rapid loss of weight. Marked abdominal distention and respiratory distress will be commonly encountered. These patients demand prompt attention to combat dehydration, toxæmia, acidosis and circulatory failure. Fatal outcome is high in this group.

Acute fulminating. The onset is usually abrupt and the progress equally rapid. The temperature tends to reach dangerous level. Vomiting is a constant and troublesome feature. Respiration is rapid and may be hyperapnœic in type. In some children the frequency ot stools may be slight or even the onset may be delayed and yet the clinical progress will be out of proportion to the number of stools. In others, stools are frequent and watery resulting in rapid loss of fluid. Dehydration, hæmoconcentration, and collapse will appear rapidly. Loss of weight which is constant may be rapid and extreme. The capillary blood count is distinctly higher than that of venous. Increased serum proteins and a moderate degree of leucocytosis are present in almost all the cases. Reduction of bicarbonates in the blood leads to a stage of acidosis. Restlessness and irritability of the early phase may soon give rise to stupor and coma. From the prognostic aspect, the appearance of acidosis, circulatory failure and the development of sclerema, are of great significance. Sclerema has been associated invariably with death; but the advent of improved methods of hydration and combating acidosis, some patients with these features have made unexpected and spectacular recovery. The progress of events is so rapid in this variety that only adequate and thorough therapeutic measures administered in good time will influence the outcome favourably.

Atypical Types. All the patients with diarrhœa do not present the usual clinical features. There are instances when patients are first admitted with the complaint pointing to other systemic disorder and only at a later stage does the true nature of the condition become evident Thus meningitic and pneumonic varieties have been observed.

Children with convulsions, neck rigidity and meningism will be admitted with the diagnosis suggestive of meningitis. But lumbar puncture will yield normal cerebrospinal fluid and only the appearance of diarrhœa, or the routine stool examination will reveal the true nature of the illness. In some of these patients herpes has been observed on the nasal folds and lips. The association of virus infection with meningitic form is very significant. Also some patients suffering from Flexner dysentery organisms have meningitic manifestations, suggesting an existence of neurotropic exotoxin. Some patients may have meningitis from other cause in addition of the diarrhœa.

There are patients in whom a sharp rise of temperature is associated with nonproductive cough, rapid pulse, prostration and moist sounds in the lungs. Respiration will be rapid and hyperpnœic, simulating acute pneumonia. Rœntgenogram examination taken within 24 hours reveals haziness in the lung fields. This type of infection has been termed the pneumonic type. Along with the disappearance of the clinical and x-ray evidence of pulmonary lesion, diarrhœa dominates the picture.

In conclusion, it may be emphasised that the child suffering from diarrhœa should be viewed *in toto*. Every effort should be made to elicit the causative factor. Stool examination, naked eye, microscopical, bacterial and cultural, should be made repeatedly as one single investigation may not yield satisfactory or convincing results.

DEHYDRATION

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Dehydration is one of the commonest and the most serious accompaniments of severe diarrhœa. Out of a series of 309 cases admitted to the Bai Jerbai Wadia Hospital for Children during the years 1949 and 1950, dehydration was present in 254 cases *i.e.*, the incidence was as high as 82·3 per cent.

The younger the child, the greater is its fluid requirements. Under the age of one year, a child requires 2½ ozs. per lb. body weight per day, whilst the adult requires only 4/5 oz. per lb. wt. per day. The daily output of fluids in infants and small children is also relatively greater, being as much as 4 per cent of the body weight per day, as compared with an output of only 2 per cent in adults. Hence it is not surprising that the incidence of dehydration is greatest in small children.

In the series mentioned, the incidence of dehydration in children under the age of one year was 69·6 per cent; between 13 months to 2 years the incidence was 17·7 per cent; from 2-5 years it was 8·5 per cent; and for cases above the age of 5 years it was only 4 per cent. Further, in the group under one year of age, 14·6 per cent were from 0-3 months old, 14·5 per cent were 4-6 months old, and 40·5 per cent were 7-12 months old. The youngest child in the series was only a day old, whilst the oldest was seven years.

The figures mentioned show, that the incidence of dehydration is greatest in the group under one year of age, specially so between the age of 6-12 months, an age when the incidence of diarrhœa is highest, not only from infection, but also from troubles arising from weaning and teething.

Prognosis

The prognosis depends greatly on the severity of the dehydration, and the time at which treatment is instituted. In this series, there was no death in the mild cases, while in the severe cases the mortality was 46·8 per cent. 35 severe cases died within 24 hours of admission, some of them even before any treatment could be started. These figures show, that quicker the treatment is started, the better is the prognosis.

Treatment

The main aims of treatment are, restoration of fluids, treatment of collapse, and the treatment of the causative condition. The latter two have already been dealt with.

Restoration of fluids: Fluids may be administered orally or parenterally subcutaneously, intramuscularly, intravenously, intraperitoneally, through the fontanelle, and by bone-marrow.

Fluids should be given orally not only as a therapeutic measure, but more important, for prevention of dehydration. The fluids that may be given orally are: (*i*) plain water. This serves to restore blood volume, renal secretion, and fluid content of the tissues in mild cases. It is by no means ideal, as it does not restore the electrolyte balance, nor does it appreciably reduce acidosis or alkalosis. (*ii*) cereal (rice or barley) water may also be used. (*iii*) whey is a bland, slightly acid fluid containing the lactalbumin, mineral salts, and sugar of milk and is readily accepted. (*iv*) saline solutions are easy to prepare, restore the Na and Cl radicles, and are readily acceptable if given in proper strength. Normal saline is often vomited, but half normal or quarter normal saline is readily taken. (*v*) Ringer's Solution (NaCl 270 grs., KCl 18 grs., $CaCl_2$ 9 grs., $MgCl_2$ 9 grs., H_2O 100 ozs.) is indicated in all forms of diarrhœa. It supplies both Cl and fixed bases (Na, K, Ca, Mg). (*vi*) sodium lactate (1/6 molar) in normal saline or Ringer's Solution is neutral and isotonic. When absorbed, it is metabolised and yields sodium bicarbonate and thus helps to combat acidosis. (*vii*) Hartmann's Buffered Lactic Acid Solution (Lactic acid B. P. 1½ ozs., 10 per cent NaOH 2 ozs., H_2O 100 ozs.) is strongly acid in reaction. This acidity inhibits bacterial growth in the upper intestinal tract. Moreover, when absorbed, the solution is metabolised and yields sodi. bicarb, and combats acidosis. However, when tried out at the hospital, it was rejected by nearly all the children. (*viii*) Darrow's Solution supplies extra potassium besides NaCl and sodi. lactate. (*ix*) Dextrose Solution given orally supplies both fluids and calories. 5 per cent solutions (1 teaspoonful in 3 ozs.) are useful; more concentrated solutions are laxative and cause troublesome distention.

Fluids that may be given parenterally: (*i*) normal saline and (*ii*) Ringer's Solution are the ones most commonly used. In the present series normal saline was employed in 221 cases. In some cases with severe dehydration and oliguria the injection of excess of these solutions may bring about retention of Cl and increase of acidosis. (*iii*) sodium lactate (1/6 molar) in water or normal saline and (*iv*) Ringer-Lactate solution (Sod. lactate 1/6 molar 60 c.c., added to 400-450 c.c., Ringer's solution) have the same advantages as when given orally. These solutions were given only to 4 cases. (*v*) Darrow found that potassium in considerable amounts was lost from the intracellular fluids *via* the stools, further aggravating the dehydration. He suggests the following solution for parenteral use: Molar sodium lactate 40 c.c., KCl 2 gms., NaCl 3 gms., H_2O 710 c.c. This solution is, however, not without danger. In moribund and babies in shock the plasma potassium may be high due to oliguria in spite of intracellular potassium deficit. For such cases Darrow's solution should only be used after renal secretion has been re-established by using other hydrating solutions. Potassium intoxication is indicated by an increase in diarrhœa and signs of heart block. In such cases 5-10 per cent calcium gluconate or calcium chloride in 25 per cent dextrose should be immediately injected intravenously. Darrow's solution was not used in the present series, but was used with beneficial results in 4 cases at the Parsee General Hospital. (*vi*) Dextrose solution supplies both fluids and calories. 5 per cent dextrose is isotonic and may be given subcutaneously. 10 or 12½ per cent solutions are best given intravenously. 25 and 50 per cent solutions are hypertonic and should not be employed alone, unless accompanied by a simultaneous subcutaneous drip of 5 per cent solution. The principle of this dual injection is as follows: the hypertonic solution given intravenously, takes up fluid from the tissues, and replenishes blood volume. At the same time the isotonic 5 per cent glucose given subcutaneously restores depleted tissue fluids. This dual injection is rarely practicable as the veins in severe dehydration are collapsed. 5 per cent glucose was employed in 21 cases in this series. (*vii*) Soda Bicarb Solution has been now given up entirely and has been superseded by sodium lactate solution which has all its advantages without its irritating properties. (*viii*) Protein hydrolysate and (*ix*) plasma supply not only fluids and electrolytes but also protein as amino acids. Protein hydrolysate was used in 15 cases as a 5 per cent solution in 5 per cent glucose or a 3-1/3 per cent sol. in 3-1/3 per cent glucose, either subcutaneously or intravenously. Plasma was used in 10 cases. (*x*) Whole blood transfusion is an excellent restorative as it supplies not only fluids, electrolytes and protein but also the cellular elements and antibodies. It should not be given in the acute stages of dehydration, because it increases hæmoconcentration. (*xi*)

Dextran Intradex-Crooks) is a plasma substitute recently put up in the market for intravenous use. I have had no experience with it so far.

Amount: The amount of fluid is to be regulated as overdosage may cause untoward symptoms.

Orally there is no limit to the amount of fluid that may be given provided that it is retained. From 30-50 ozs. or more may be given, the ideal to be aimed at being 3-4 ozs. per lb. of body wt. per day.

Parenterally 60 c.c. per lb. of body weight per day is a safe amount. However, at any one time the amount of fluid injected should not exceed 10 c.c., per lb. of body wt. For children under the age of 2 years the amount of fluid run in at any one time should not exceed 250 c.c.

The amount of fluid and the number of injections per day vary considerably, depending on the severity of dehydration. For example, in this series a mild case received only one injection of 100 c.c. of subcutaneous saline per day for 2 days, whilst a very severe case received 150 c.c., 4 hourly for as many as 25 days, before it recovered. Besides normal saline this particular case received plasma and whole blood.

The untoward signs of superhydration that may be met with are pulmonary œdema and œdema of the skin and subcutaneous tissues. Oliguria occurs when excess of saline solution has been given. None of the cases of the present series showed any signs of superhydration.

Technique: Oral fluids may be given freely if vomiting is absent. In very mild cases oral fluids only are sufficient. There were 10 such cases in the series. If vomiting is present, fluids are best given cold and in small sips only, every few minutes. Should this method also provoke vomiting, fluids should be given by catheter. The technique is as follows: The distance from the bridge of the nose to the tip of the xiphisternum is measured with the baby supine, and ½-1" added to this length. The baby is now put up in a semi-sitting position and a catheter approximately the thickness of the baby's little finger is passed down the mouth or nose for this length. This measurement is important, because at this length the tip of the catheter is just within the cardia. If pushed beyond this length, the catheter tends to coil up in the stomach and provokes vomiting. After inserting the catheter, fluids are run in, in small quantities (5 c.c.,) by syringe, or the catheter connected to a drip apparatus and fluid allowed to drip in at a rate of 20-40 drops per minute. When withdrawing the catheter, care should be taken to hold it firmly pinched to avoid spilling of fluid into the trachea. A preliminary stomach wash helps greatly in promoting retention of fluids. For this purpose the catheter is pushed 3-5" further down, the washing completed with saline and fluids run in after withdrawing the catheter to the proper length. Intragastric drip was employed in 14 cases of this series.

Parenteral Fluids: The subcutaneous route is the one most commonly employed. It was used in 224 cases. The site of election for injection is the outer part of the thighs. The abdomen and axillæ are best avoided as they cause respiratory embarrassment from pain. The needle chosen should be a wide-bore one (20 gauge BD), the subcutaneous tissues well pinched up, and the needle inserted deep into the subcutaneous space. Both thighs should be used simultaneously. The intramuscular route may also be employed. The technique is similar, the muscle employed for this purpose being the vastus lateralis. By both these routes fluids may be run in by drip at a rate of 20-40 drops per minute. When from lack of facilities a syringe has to be used, it should preferably be a big one (20 or 50 c.c.), or it should be fitted with a two-way stopcock, one end of which is connected to the needle and the other to a tube immersed in the injection fluid. The injection is given slowly, and the position of the needle changed from time to time as soon as a swelling of some size appears. The fluids that may be injected by these routes are any of those previously mentioned for parenteral use except hypertonic glucose and blood.

The chief difficulties met with when giving fluids by these routes are induration and pain. These can be avoided by adopting proper technique, and the use of hyaluronidase (Hyalase—Benger). Hyalase greatly facilitates the spread of fluid in the tissues and cuts down the absorption time One ampoule contains 1,000 Benger units (*i.e.*, 1 mg., of Benger standard hyaluronidase fraction derived from 0·5 gms., of fresh mammalian testis), and is sufficient for the infusion of 500-1,000 c.c. of fluid. This is the optimum quantity of hyalase required, and addition of more hyalase will not further increase the rate of absorption. Lesser amounts may be used for the subcutaneous administration of smaller volumes. When given by drip the best method is to let some 10-15 c.c. of fluid run in and then inject the hyalase dissolved in 1-2 c.c. of water into the rubber tubing close to the needle. When giving fluid by syringe, hyalase may be incorporated in the injection fluid, but care should be taken to use up the fluid as soon as possible, since hyalase deteriorates quickly in solution. The use of hyalase facilitates frequent injections at the same site. In some cases, however, it has failed, and badly too—the site remaining painful, indurated and red for as long as 3-4 days.

The intravenous route is always difficult in small children, especially so in dehydration when the veins are collapsed. Dissection of the veins had often to be resorted to; but with the advent of Hyalase this technique is not as necessary now as before. The veins that may be used for injection are the antecubital, the femoral, the saphenous vein, the external jugular, and the scalp veins. The rate of drip should not exceed 30-40 drops per minute, any of the fluids mentioned previously may be employed. This route was employed in 16 cases.

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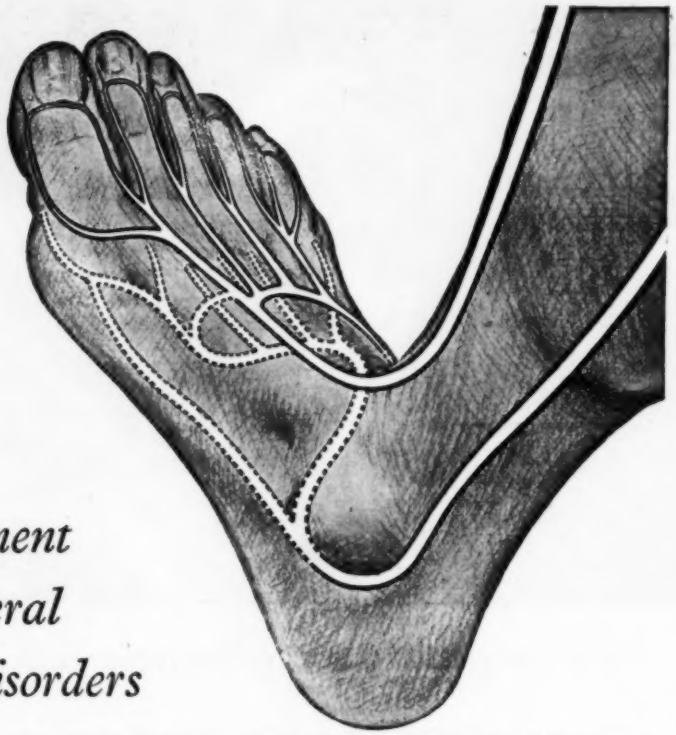

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Fontanelle puncture may be used by the experienced physicians for the introduction of non-irritating bland fluids *e.g.*, normal saline, plasma and blood. Glucose should never be given by this route. The head is shaved and the baby held tightly supine by an attendant, with the head and trunk in a straight line. The longitudenal sinus is now punctured by inserting a 3/16" 20 gauge BD needle perpendicularly, exactly in the centre at the posterior angle of the fontanelle, applying suction on the syringe throughout, till blood is withdrawn. The fluid is now slowly run in, care being taken not to move the needle. The chief danger of this procedure is transfixion of the sinus, with consequent injection of the fluid into the brain tissues resulting in convulsions. Blood was given to 3 cases by this route.

The intraperitoneal route is an easy and safe one. Only bland non-irritating fluids should be used. Dextrose should never be employed. The bladder should be empty. A not too sharp 20 gauge BD needle is introduced at a point mid-way between the umbilicus and the symphysis after pinching up the abdominal wall. The bevel of the needle should face the symphysis. The needle is shoved in upto a point when a 'give' is felt. As soon as this occurs the direction of the needle is changed, it now being directed upwards and in a plane parallel to the abdominal wall. The needle is thrust in upto the hilt. Warmed fluid is now run in at a fairly quick rate either by drip or by syringe until the abdomen begins to distend. The average quantity required is 150-250 c.c. Contraindications to this route are respiratory complications, gaseous distention of the abdomen, vomiting and peritoneal irritation. This route was used in 4 cases.

I shall not speak here in detail of bone marrow infusions, as they proved to be both difficult and unsuccessful even with special apparatus. The tibia is employed in children under the age of 7 years, the sternum for children above this age. Blood was given to 2 cases only by this route, and had to be pushed in with great difficulty by syringe.

TABLE 1—INCIDENCE OF DEHYDRATION.

Total No. of Cases ...	309	
Cases without Dehydration ...	55	17·7%
Cases with Dehydration ...	254	82·3%
Severe Dehydration ...	152	59·9%
Moderate Dehydration ...	102	40·1%

TABLE 2—AGE INCIDENCE.

Under one year	177	69·6%
0-3 months	37	14·6% of total
4-6 ,, ...	36	14·5% ,,
7-12 ,, ...	104	40·5% ,,
13-24 Months	45	17·7%
25 Months—5 years ...	22	8·5%
Over 5 years	10	4 %

TABLE 3—SEX INCIDENCE.

Males	146	57·5%
Females	108	42·8%

TABLE 4—MORTALITY.

	Total	*Expired*	*Improved*	*D.A.M.A.*	*Cured*
Moderate Cases ...	102	0	28	8	63
	%	0	27·5	7·8	61·8
Severe Cases ...	152	71	16	3	62
	%	46·8	10·5	1·9	40·8

Died on Admission ... 35 cases.

TABLE 5—FLUID ROUTE.

No oral fluids	15 cases
Oral fluids only	10 ,,
Parenteral Fluids: Subcutanous ... Intramuscular ...	... 224 ,,
Intravenous	21 ,,
Intraperitoneal	4 ,,
Fontanelle	3 ,,
Bone Marrow	2 ,,

TABLE 6—PARENTERAL FLUIDS USED.

Normal saline	221 cases
Ringer's Solution	— ,,
Dextrose solutions	21 ,,
1/6 Molar Sod. Lactate	4 ,,
Darrow's Solution	— ,,
Plasma	10 ,,
Protein Hydrolysate	15 ,,

DIAGNOSIS AND PROGNOSIS

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I shall try to say a few words on diagnosis, differential diagnosis, and prognosis of diarrhœa in children.

In some cases, the diagnosis requires very little work. In certain cases, further studies will have to be made, while in others, the answer will be apparent early in the course of investigations; occasionally, the case will remain an enigma even after the most extensive work. Diagnostic procedures include complete history, complete physical examination, stool examination if fresh stools are available. This again includes gross examination by physician, guaiac test for occult blood, microscopic examination for ova, parasites, cyst, and vegetative forms of amœba; rectal swab examination is useful when fresh stool is not available. Examination of total white blood corpuscles and differential count is done in every case of diarrhœa.

When a case is brought with a complaint of frequency of loose motions, a careful case history is essential in arriving at a diagnosis as to the type of diarrhœa. The laboratory examination will later on confirm the diagnosis. The history helps much. It must include as far as possible the approximate date of onset of the complaint with a note of associated circumstances. The patient's relative should be asked about what he means by diarrhœa. Because the definition of diarrhœa in his mind may not coincide with that in the physician's mind. A mere statement that the child has diarrhœa should not be taken on its face value. The case history must, therefore, include number and character of stools, whether they are constant or intermittent, whether the stools contain blood and mucus, any curds, or undigested material, duration and progress of the complaint, the mode of onset, whether the diarrhœa started with feve. or that fever and vomiting preceded the onset of diarrhœa, whether it is associated with abdominal discomfort or pain and the location of the pain.

History is followed by careful physical examination. General appearance of the child is very important. This will give you an idea of the nutritional state of the child. Signs of deficiency or malnutrition are important causative factors and also the result of diarrhœa. Careful palpation of the abdomen and then auscultation are important procedures in these cases. The examination of other systems as a routine is also very important.

The history and physical examination will help us in knowing whether the diarrhœa is infective, or non-infective. If infective, we must find out by complete physical examination whether it is enteric, local and primary; or non-enteric, parenteral and secondary. History of an epidemic is very important. History of fever, cold, cough, pain in the ears or ear discharge, a boil etc., any extra-enteric infection is very important. If we get the history of fever and vomiting preceding the onset of diarrhœa, infection should be suspected. The infection as the cause of diarrhœa is again confirmed from the history that the food is suitable for the child, that the food is given at proper intervals and has been agreeing with the infant. Thus the history alone may help us in coming to a conclusion that the diarrhœa is due to some extraneous factor most likely a parenteral infection. In order to confirm the diagnosis, careful search should be made for the presence of infection. Examination of the skin for the presence of any furuncle or injury, examination of the ear, nose and throat and the examination of other systems are made The latter when diseased, give leading symptoms of the system affected. But at times, the attention of the physician is diverted by the diarrhœa and the real cause is overlooked. Therefore, one cannot depend every time solely on case history and physical examination, but one must require the help of laboratory report. Climate and economic conditions play an important role in acting as a predisposing factor. In our country, we see increased number of cases of diarrhœa in infants and young children during hot weather. More or less the same incidence is seen in America and Australia. The incidence is much less in British Isles. The diagnosis of the parenteral diarrhœa is thus based on the finding of an extra-enteric focus and negative stool culture. The diagnosis of diarrhœal condition due to an unknown cause, is a difficult problem. Here the primary site of infection and etiologic agent is not known and therefore cannot be localised. We do not know whether the diarrhœa is enteral or parenteral with a secondary extension to the bowel. In such cases, the age of the child (usually under one month), epidemicity, character and course of the disease, will be useful in diagnosis. Stool culture is negative. Confusion of the diagnosis will occur, when attempts are made to appraise the individual case, or the group of cases, apart from its epidemiological settings. This difficulty of acute diarrhœal diseases of the new-born infants offer a challenge to the diagnostic acumen of practising physicians.

If high temperature is coincident with the development of diarrhœa, and if no parenteral infection is discovered, one may suspect bacillary dysentery, especially Shiga type. But an absolute diagnosis cannot always be made early in the course of the disease. Later, the appearance of blood, pus and mucus in the stools or the findings of dysentery bacilli on stool culture, may make the diagnosis quite clear. The complaint of tenesmus, pain in the abdomen, restlessness and anorexia will be always available in such cases. The problem of enteral infections is covered mainly by dysentery bacillus which is more common cause of enteral infection than entamœba histolytica. From a practical clinical standpoint,

differential diagnosis chiefly concerns acute appendicitis, pneumonia, meningitis, amœbiasis, typhoid, salmonella or staphylococcal food poisoning, and focal non-specific enterocolitis. A positive stool culture, a rising agglutination titre and a purulent cytology of the mucosal exudate constitute a final diagnostic tetrad in laboratory diagnosis of acute bacillary dysentery. Salmonella infections and typhoid give positive blood and stool culture, a rising agglutination titre, and no response to sulphonamide. Wet smear examination for demonstration of trophozoites, annd stained smears for cyst will give us diagnosis of amœbic dysentery. In Bombay, some cases of diarrhœa are due to Giardia lamblia and Balantidium coli. Stool examination helps in such cases. Ingestion of food, poisoned by toxins produced by some varieties of staphylococci may precipitate sudden onset of gastro-enteritis. History of number of people getting the attack, after eating the same food, mode of onset and short duration will be helpful. Same picture is given by ingestion of heavily polluted water, inorganic toxic products, drugs and animal poisons. These cases simulate cases of bacillary dysentery. Other confusing but less frequent diseases are intussusception, influenza, Henoch's purpura, rheumatic abdominalis, intestinal parasitism, adenomatous polyp of the colon and malaria. All these can be ruled out by careful clinical and laboratory studies. There is another variety in which the diarrhœa is due to organisms whose pathogenicity is questionable. No specific pathogens are found. Common organisms found in the stools of such patients are, Proteus morgagni, Bacillus pyocyaneous and late lactose-fermenting members of the colon-aerogenous groups. Only laboratory finding will be helpful to differentiate these from mild shigella infections. In diarrhœas due to virus, there is no satisfactory laboratory animal available for virus studies. Intestinal influenza also gives rise to gastro-intestinal symptoms, namely nausea, vomiting and diarrhœa with fever etc. Blood examination shows leucopenia.

Before going to consider non-infectious variety, it will not be out of place, if I try to describe in a few words about the investigations, namely, naked eye and microscopical examination of voided stools and rectal swabs, the examination of blood and urine. We must see the consistenry of the stools, colour, smell, presence of mucus and blood, curds, etc. This is followed by microscopical examination which helps us in differentiating the diarrhœa due to enteral infections from other types. Stool culture will show the offending organisms when the diarrhœa is due to enteral infections. The offending organisms may be dysentery bacillus, various atypical strains of colon bacilli, number of types of streptococci, the bacillus mucosus, bacillus pyocyaneus, shigella group of bacilli and a large group of closely related organisms exist which are capable of causing enteritis. The bacteriology of the stools does not differ from that of normal infants, in the diarrhœas due to parenteral infections and to unbalanced diets. Stool culture is not however practicable in every case. As for the examination of the voided stool sample, there are many difficulties and they are:—

(1) in getting fresh sample of stool,
(2) in getting a sample of stool because of ignorance of parents.
(3) samples are brought but they are either dry or mixed with urine.

All these can be overcome by examination of a rectal swab. This is a very easy and handy method, which can be carried out by each and every physician. The advantages are that you get a fresh sample of stool for examination, no trouble or strain for parents who usually show unwillingness in collecting and bringing of the sample and there is less chance of interchange of stools. A proctoscope with 3/4" diameter, glass slide, coverslip, normal saline, iodine solution, soft paraffin and a broom stick to which a cotton swab is attached, are the things needed for the rectal swab examination. For grown-up children left or right lateral position and for infants, it should be kept on mother's laps with the face of the infant downwards. The film made in saline and iodine solution is examined under low and high power. Coelho of B. J. Hospital, Bombay, has successfully proved that the results of the rectal swabs are more reliable than those of voided stools in finding the following: macrophages, pus cells, amœbæ—both vegetative and cyst forms, and giardial infestations; while rectal swab examination is less helpful in finding rodnd worm, thread worm and whip worm ova. Here the voided stools are most useful.

The examination of the blood will point to the presence of infection.

The examination of urine will help us exclude any disease of the urinary system. We have noted in B. J. Hospital that pyelitis is a less common cause of diarrhœa than either the upper respiratory tract infection or otitis media.

After excluding the infective variety, I shall try to differentiate the non-infective group of diarrhœas. Before describing the more common varieties, I shall finish in a few lines some very few and uncommon conditions in which diarrhœa is a common complaint. Subacute or acute yellow atrophy of the liver, cœliac disease, and diarrhœa in a nervous and hypertonic child. In the hypertonic child, the threshold of nervous impulses is lowered. Response to any irritation answers in an exaggerated manner. Every food is followed by stools, and colic in between. Diarrhœa associated with teething is explained on the ground that nervous heredity, rickets and spasmophilia are more responsible and the process of dentition lights the fuse. More important group in non-infective variety is due to dietetic error. In this, we usually find from the history that the composition of the feeds is not suitable,

there may be deficiency or excess of any one of the constituents or usually there may be frequent and irregular feeding. In some there is too early addition of rice and rice flour. Underfeeding due to much dilution of milk may precipitate diarrhœa when the child has reached a state of severe malnutrition. Many a time it is very difficult to know from the history whether the child is overfed or underfed as the history given by the mother is quite incomplete and unreliable. However, when history of overfeeding of an unsuitable milk mixture is available, it helps much to make alteration in the mixture. Excess of fat in the feeds precipitates diarrhœa showing some neutral fat, soaps, or fatty acids in the stools. The stools are offensive. When there is excess of carbohydrate, the stools are acid in reaction and sour-smelling. Insufficient addition or too much addition of sugar causes disturbance in digestion of fats and proteins. Indigestible food substances also produce frequency of stools, but it is a temporary phenomenon. Decrease in the gastric juice as seen in marasmic children, premature babies, convalescent children after an acute illness or some chronic debilitating disease and with severe anæmia, causes digestive disturbances. These subjects also become easy victims of infection either extraneous or that infection from the lower bowel ascends up and precipitates diarrhœa. Deficiency of vitamins, A, B, D singly or in combination causes digestive disturbances. Signs of vitamin deficiency should be carefully examined. In some case, we get the history of sensitivity to certain foods. In very rare cases a child is sensitive to mother's milk. In some cases, sensitivity to cow's milk is seen. Change in the type of milk will set the things right. Lastly, before concluding the talk on diagnosis, one more condition should be kept in mind for differentiation, *i.e.*, nervous manifestations. They are associated with diarrhœa and dehydration. The condition simulate organic disease. In case of doubt, a lumbar puncture is done.

Thus it will be seen that careful history taking, complete physical examination, examination of the rectal swab, urine and blood will help us much to come to a diagnosis.

Prognosis

This depends upon the general state of the child, age, epidemicity, the treatment when it is started, the virulence of infection, duration of illness etc. Prognosis in epidemic diarrhœa of the new-born is very poor. Signs of severe dehydration namely depressed fontanelle, sunken eyes and temporals, ingoing of cheeks with prominence of cheek-bones and nose, dry lips and viper tongue, distention of abdomen, intractable vomiting, high temperature, drowsiness, convulsions, restlessness, sclerema etc., when present in combination, foretells bad prognosis. The present-day clinical study, advanced knowledge of various means of finding out the cause of diarrhœa, knowledge of hygiene in the feeds and feeding utensils of the child, advanced knowledge in dietetics of the child including early administration of vitamins and other deficiency constituents according to the needs of the individual child, the knowledge in the means to remove dehydration, the more and more use of chemotherapy, penicillin and now streptomycin and aureomycin according to the etiological agent, all these means have greatly changed the prognosis of diarrhœa. In our country, because of ignorance, overcrowding, unhygienic living etc., the percentage mortality due to diarrhœa is still high.

TREATMENT

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For simplicity and convenience, we shall discuss the treatment in the following order:—

(1) Correction or dehydration and resulting acidosis and shock.

(2) Control of infection—enteral and parenteral meaning those in the alimentary tract and outside the alimentary tract respectively.

(3) Dietetic measures.
(4) Symptomatic treatment.
(5) Miscellaneous medicinal therapy.
(6) Supportive measures during the acute phase and convalescence.

Correction of Dehydration

Mortality is very high in children with severe dehydration, specially so in infancy. A prompt treatment of dehydration and preferably prevention of severe types should be considered as the most important of all the therapeutic agents. This has been already discussed.

Control of Infection

(a) Enteral Infection:

1. *Common non-specific group—of doubtful etiology*—Abnormal bacterial flora in the bowel may be exogenous *i.e.*, introduced from outside or endogenous meaning upward spread of the normal lower bowel organisms specially B. coli to upper bowel which is usually sterile. *Virus* has been thought of as a cause in some cases.

2. *Specific group*—Specific infections may give rise to diarrhœa and vomiting, and in some cases, may not develop their characteristic clinical picture at all.

Bacillary dysentery group is the most frequent; the others being giardiasis and worms. *Amœbic infection, typhoid and salmonella* infection, though rare, should be remembered as occassionally they do cause diarrhœa even in young infants. Dr. Coelho has observed a case of diarrhœa due to amœbic infection in a fifteen days' old baby. I have seen three cases

of typhoid fever in young infants presenting a picture of fever and diarrhœa only. Cholera should be remembered as a specific cause of diarrhœa and vomiting during an epidemic.

(B) PARENTERAL INFECTION:

Upper respiratory tract infection *viz.*, rhinopharyngitis and tonsillitis seem to play an important role in acute diarrhœa, as a number of cases specially infants develop diarrhœa following an upper respiratory tract infection. The parenteral infection should be searched for and treated adequately as in many cases, the diarrhœa and vomiting will not be well controlled as long as the infection is active.

Supha Drugs—Diffusible sulpha drugs like triple sulpha or sulphadiazine are routinely used by us.

Dose—Total daily dose is 1 to 1½ gr. per lb. of body weight to be administered in 4 divided doses. The initial dose being double the maintenance dose, *e.g.*, in a child weighing 15 lbs., the initial dose is one tablet followed by half tablet every 4 to 6 hours. This is to be continued for two days after the stools are well formed. Usually the drug should not be used longer than 5 to 7 days. Children can tolerate large doses of sulpha drugs and the practice of giving small and ineffective doses should be deprecated. Vomiting usually due to infection is not a contra-indication to sulpha group of drugs and in most of the cases it subsides with adequate treatment of infection. When vomiting persists antibiotics are resorted to. Triple sulpha is preferable when there is severe dehydration and oliguria.

In our experience diffusible sulpha drugs have always given better and quicker results than non-diffusible ones like sulphaguanidine, sulphasuxidine and sulphathalidine.

Advantages of diffusible sulpha drugs are probably due to (1) their quick and effective penetration in the lesions in the wall of the gut, and

(2) Their action on the parenteral infections. Non-diffusible sulpha have only local action in the lumen of the bowel.

Results—The stools are usually formed within three days of sulpha therapy in the majority of cases, while the fever touches normal within 24 to 48 hours.

Contra-indications—Diffusible sulpha are contra-indicated whenever there is severe anæmia, jaundice, marked oliguria, and when there is a definite history of allergy to sulpha drugs. In these cases antibiotics should be used.

Whenever a child does not respond well to sulpha drugs within 4 to 5 days—(1) search for a parenteral infection which cannot be effectively controlled by sulpha alone; (2) when the diarrhœa is due to organisms like B. proteus, B. pyocyaneus which are not sensitive to sulpha, or the diarrhœa may belong to a specific group *e.g.*, amœbic infection, typhoid etc.

Sometimes the diarrhœa is not checked effectively because there is some neglect in the dietetic control—most common in our experience are (1) that the bananas are not given in adequate quantity; (2) the mother has been feeding the child with milk right from the beginning.

Antibiotics are very valuable in the treatment of diarrhœa in children.

Penicillin should be used in addition to sulpha drugs for quicker and better control of parenteral infection specially in serious cases.

Dose—1,00,000 to 2,00,000 units of fortified procaine penicillin can be injected intramuscularly once or twice a day depending upon the severity of the infection.

Streptomycin is usually used in cases where sulpha drug is contra-indicated or when the infection does not respond to sulpha drug alone.

Route of Administration—It can be given orally for its local action alone, or oral and intramuscular therapy may be combined when parenteral infection or a complication is suspected.

Dose—100 mgm. every 3 to 4 hours can be given to a year old child. It can also be added to the fluids like Ringer's solution or glucose saline which are given frequently in small quantities.

Advantages—It is better tolerated in presence of vomiting.

Disadvantages—rarely after initial improvement due to eradication of sensitive organisms, a relapse may ensue due to unchecked activity of streptomycin-resistant organisms.

Results—Conflicting results are reported by James, Holzel, Magnussan and others. In some cases this improvement is spectacular, the stools, toxæmia and fever responding within 2 days and the milk feeds can be started within 24 hours with quick gain in weight. In some cases it fails completely. In individual cases the results of streptomycin therapy are unpredictable.

Aureomycin—This antibiotic has a unique position because of its broad antibacterial spectrum being effective on gram-positive and negative organisms and some viruses. The high concentration of the drug in the blood, and the fæces is a further advantage in controling both enteral and parenteral infections. Magnussan and others have reported good results in eight infants with diarrhœa and vomiting. These cases were resistant to sulpha, penicillin and streptomycin therapy but made striking clinical recovery with aureomycin, administered in a dosage of 25 mgm. every 4 hours.

I have used aureomycin spersoid in the dosage of 25 mgm. every 6 hours for 5 to 7 days with good results. Recovery was spectacular in a 15 days' old baby admitted with severe diarrhœa, dehydration and shock. Within 48 hours of aureomycin administration, the child improved remarkably and was discharged in excellent

condition in a week. In another case the child who did not respond to a combined sulpha and streptomycin therapy for a week, improved within 24 hours of aureomycin administration.

Chloramphenicol (Chloromycetin)—Rogers and others in a preliminary report in B.M.J. has noted most encouraging results with Chloramphenicol in an epidemic of infantile diarrhœa and vomiting. In all the cases, Bacillus coli B.G.T. was found on stool cultures. The cases were resistant to sulpha, penicillin and streptomycin both clinically and bacteriologically, but showed both clinical and bacteriological recovery within 24 to 48 hours of therapy. The daily dosage required was 75 mgm. per lb. body weight and was administered in 6 to 8 divided doses, the initial dose being twice the maintenance dose. I have little personal experience of this drug in the treatment of diarrhœa. The high cost and the large dosage required (six capsules in a day for a child weighing 20 lbs.) prohibit its use in most of the cases. Aureomycin is cheaper and effective in smaller doses.

Terramycin—The results of this new antibiotic of a wide range of action will be eagerly awaited.

High concentration in blood and fæces will increase its scope for treatment in diarrhœa in children while availability in a stable solution form will be an ease in administration.

Symptomatic Treatment

1. *Vomiting*—Vomiting is usually due to infection both enteral and parenteral. A prompt and adequate treatment of these infections with chemotherapy and antibiotics will control it in the majority of cases.

Oral streptomycin in a daily dose of 500 to 800 mgm. added to total daily requirements of fluids like glucosaline or Ringer's solution and administered frequently in small amounts, is very useful in stopping vomiting. In these cases it acts probably by eradication of abnormal bacterial flora in the stomach and upper bowel.

Complete starvation for a period of about 12 to 24 hours and *parenteral* administration of glucose and saline will relieve the vomiting in the majority of the cases.

A stomach wash with normal saline may have to be resorted to in some cases where vomiting is foul-smelling or contains a lot of mucus.

Later in the course of the disease vomiting is usually due to faulty technique of feeding which causes overdistention of the stomach with air. This should be corrected by proper attention to the technique of feeding. But persistent vomiting should warn us of complicating infections.

Drugs—Acid hydrocyanic dilute ¼ to ½ min. per dose with bismuth mixture can be given but has a limited value.

Phenobarbitone ¼ to ½ gr. before feeds may stop the vomiting of a restless child.

Feeding should be introduced cautiously in small amounts and diluted skimmed milk formula will be preferable. Neglect may restart the vomiting.

2. *Fever*—It is due to infection, dehydration or both, and so this should be adequately treated.

Antipyretic drugs—Acetylsalicylic acid ¼ to 1 gr. for a year old child may be used for high fever.

Hyperpyrexia should be treated promptly by cold wet packs.

3. *Griping pain in the abdomen and tenesmus*—Tincture opii ¼ to 1 min. per dose can be given to a child one year old. Tincture opii is contra-indicated if there is distention of the abdomen, drowsiness, and severe toxæmia. It should be avoided in young infants.

Tincture opii camphorata 5 min. per dose can be given orally in infants.

4. *Excoriation of the buttocks and anal region and at times scrotum and heels.*

This is due to frequent irritation by acid stools. Mechanical trauma by improper cleaning worsens the condition. Early attention can prevent this condition.

(1) Zinc and castor oil paste for external application to the affected parts.

(2) The affected parts should not be cleansed with a dry cloth. The parts should be cleansed with warm oil. Moist cotton wool should be used for dabbing the part.

(3) Gentle colonic lavage with a warm saline solution reduces the frequency of exhausting acid stools.

(4) Protein milk diet is most valuable as it produces alkalinity in the colon.

5. *Distention of Abdomen*—If moderate, it is usually due to enteritis itself, or by air-swallowing from faulty feeding.

Carbachol tablet (2 mgm.) ¼ tablet for a year old child can be given as required. In resistant cases carbachol or pitressin injection ¼ c.c. subcuteneously can be injected 6 to 8 hourly.

Turpentine stupes may be helpful in some cases.

6. *Paralytic Ileus*—This is a dangerous complication of diarrhœa. The treatment consists of—(1) continuous aspiration of the stomach with a Ryle's tube. (2) Carbachol, pituitrin or prostigmine injections in a dosage of ½ c.c. for a child one year old injected every 6 to 8 hours. Simultaneously a flatus tube should be introduced into the rectum for about fifteen minutes. With early and adequate control of infection this is a preventable complication in most of the cases.

7. *Restlessness and Irritability*—These are usually due to dehydration or high fever, which should be attended to promptly.

Barbiturates like Gardenal ¼ to ½ gr. or Seconal ½ gr. can be given to one year old children and repeated, if necessary.

8. *Convulsions*—Convulsions during a course of acute diarrhœa can be due to any of the following:

(1) High fever, (2) dehydration, (3) toxæmia, (4) meningeal infection, (5) rarely thrombosis of superior longitudinal sinus, (6) Guanidine intoxication and (7) tetany of alkalosis in rare cases.

Lumbar puncture should be done to exclude meningeal infection specially so when the anterior fontanelle is bulging.

Calcium gluconate 5 per cent 5 c.c. can be injected intravenously for tetany.

Seconal sodium—A capsule of ¾ gr. can be introduced into the rectum after making a few punctures in the capsule with a needle, so that the drug is quickly absorbed. The buttocks should be strapped for about fifteen minutes otherwise the capsule may be thrown out.

9. *Diminution of urine or anuria* is usually due to severe dehydration which causes diminished blood volume, circulatory failure and ineffective glomerular filtration. The dehydration should be adequately treated by parenteral fluid therapy.

Rarely anuria or oliguria persists in spite of correction of dehydration. This is probably due to toxic damage to the kidneys. One of our cases of anuria did not respond to any measure including sodium sulphate 4·2 per cent intravencusly.

10. *Shock* should be promptly and adequately treated. A number of cases are brought to the hospital in the condition of shock. It is usually due to dehydration but rarely it can be a result of overwhelming toxæmia specially in cases of severe bacillary dysentery in which the child may die of toxæmia before a severe diarrhœa can develop.

The following measures should be instituted:

(1) Warmth—The child should be kept in warm surroundings.

(2) Oxygen—By intranasal catheter though oxygen tent is preferable.

(3) Analeptics—Coramine ½ to 1 c.c. for a year old child every 4 to 6 hours.

(4) Plasma—10 to 15 c.c. per lb. body weight intravenously.

(5) Adrenal cortex preparations—Rapidly acting watery preparations like Eucortone or Cortin is preferable. Dose is 2 c.c. I.V. with parenteral fluids followed by 1 to 2 c.c. I.M. every 6 to 8 hours.

11. *Dyspnœa* is due to acidosis which should be treated with 1/6 molar solution of sodium lactate.

Penicillin or aureomycin should be used if there are bronchitic signs in the lungs.

12. *Hæmorrhages*—(1) Vitamin K 10 mgm. should be given by I.M. injections; (2) Rutin 10 mgm. thrice a day orally; (3) A blood tranfusion is indicated.

Miscellaneous Medicinal Treatments

1. *Cathartics*—These are contraindicated in most cases. As the infant is usually seen after the diarrhœa has continued for some time, no good is to be accomplished by the use of a cathartic. It should never be given if there is any dehydration as it will aggravate it and may precipitate the end. Routine use of castor oil should be deprecated.

2. *Opium preparations* have been discussed.

3. *Bismuth, chalk and kaolin preparations* are commonly used but to be effective, they should be given in large doses. Bismuth carb 15 to 30 grs. per dose should be given to a year old child, and repeated every four hours. It should be omitted gradually within a week. Bismuth subnitrate should not be used at all as reduction of nitrate into nitrites by intestinal bacteria may result in nitrite poisoning.

Kaolin and pectin mixtures when used should be in large doses *e.g.*, kaolin in 1 to 2 drachm doses every two hours till the stools are formed and toxæmia improved.

Antihistamines—They have been recently introduced in the treatment in diarrhœa. Though the exact nature of products responsible for the causation of symptoms is not known, histamine and other amines have been assumed to be the toxic agents. Histamine increases the capillary permeability of intestinal vascular plexus with outpouring of serous fluid resulting in diarrhœa. Antihistamines have been used with an idea to neutralise the histamine. In a priliminary report Neumann has reported with control studies, that the group of cases receiving antihistamines showed quick improvement in stools and toxæmia. I have used them in a few cases but it is too early to form an opinion.

Supporting Measures

Blood Transfusion—Blood transfusion is very helpful—1. in cases of diarrhœa where the infection is persistent, 2. in shortening the convalescence of severe cases, 3. in cases associated with severe anæmia and malnutrition. It is usually not necessary in cases where the child's previous nutrition was good.

Blood transfusion should never be given unless the initial dehydration is corrected by other fluids. The best time for the first transfusion whenever indicated, is 12 to 24 hours after the restoration of fluid balance.

Convalescence—Crude liver extract and vitamin B complex preferably by injection and dilute hydrochloric acid and protein hydrolysate orally should be given in cases where diarrhœa has been severe or prolonged and in those with under-nutrition, anæmia and avitaminosis.

As definite liver damage (fatty degeneration and necrosis) has been found in cases of severe or prolonged diarrhœa, these supporting measures will help in quick improvement of the liver condition. Neglected cases often present a picture of anorexia, poor digestion, distended abdomen and liver enlargement following an attack of diarrhœa. These cases are not infrequently met with in practice.

SPECIAL ARTICLE

CHRONIC FLUORINE INTOXICATION —A REVIEW

PART II

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(*Continued from previous issue*)

In Part I of this review, the possibilities and symptoms of acute intoxication by fluorides was dealt with[1]. This part reviews the present state of our knowledge on the mechanism by which these symptoms are caused and discusses the methods available for their amelioration.

Mechanism of Production of The Symptoms

Mottling of the tooth enamel—Mottling of the tooth enamel is believed to be caused by a direct effect of the fluorides on the enamel-forming cells and not by an indirect effect exerted through the levels of calcium and phosphorus in the blood[2]. Dietary supplements of calcium, phosphorus or vitamins do not help. Mottled enamel can occur only among those children who are exposed to the influence of fluorine during the period of tooth-formation and the condition is irreversible in man and such animals whose enamel-forming mechanism disappears in later life. Hence the only method of preventing this symptom is the removal of fluorides from the water.

Mechanism of Skeletal Changes

Bone composition—The mechanism by which these abnormal changes are produced in the skeleton is not quite clear yet. The bone undergoes changes both in structure as well as in chemical composition as a result of taking fluorides. The major portion of the fluorine retained by the body is stored in the bone which may show considerable increase in this constituent long before other symptoms manifest themselves. It has also been recorded that the distribution of fluorine in all the bones is not uniform, the vertebræ and cancellated bones showing a comparatively higher proportion of fluorine.

Published results of variations induced in other ingredients such as ash, calcium or phosphorus are very conflicting. Lantz and Smith[3] suggest that this conflict is due to the fact that these constituents are greatly influenced by the diet and age of the experimental animal. The content of magnesium and carbon dioxide have been claimed to be more faithful indices of fluorine intoxication[4, 5].

Structural Changes

Both osteoporotic and osteosclerotic changes have been noticed in animals receiving fluorides. Here again, the diet, the age of the animal and its state of calcium requirement probably play a part.

Antagonistic Action of Fluorine

Histological studies afford evidence of considerable withdrawal of calcium from the skeleton. According to Roholm[6], such withdrawal is due to the "calcioprive" action of fluorine and is probably caused by the imperative need to detoxicate the fluorine rapidly. The calcium excreted through the fæces is, however, not increased. This puzzle is sought to be explained on the suggestion that the calcium so mobilised from the skeleton is immediately re-deposited as bone-like tissue. It is suggested that fluorine has two antagonistic actions. While it withdraws calcium from the bone, it stimulates the formation of new bony tissue[7]. Yamane[8] found that sodium fluoride augments the activity of bone phosphatase. Phillips[9] found an increased serum phosphatase in calves receiving fluorides. Finn and Kramer[10] found that the administration of fluorides helps fractures to heal more quickly.

Though bone formation is stimulated by fluorine, it would appear that the newly-formed osseous tissue is abnormal in structure. Calcification is abnormally high and the calcium salts are deposited irregularly in coarse granular lumps.[11] Early workers suggested that these coarse deposits were crystals of calcium fluoride but Roholm[6] showed that this view was erroneous.

Thus fluorine appears to withdraw calcium from normal bone and cause it to be redeposited irregularly and in atypical forms. Why the bone formed under the influence of fluorides is atypical, in what respects it differs chemically and physically from normal bone and why calcification occurs in cartilaginous tissues and why certain areas are preferred for such calcification are the key questions for which answers have to be sought. Majumdar *et al*[3] have shown that the exostoses on the long bones contain a larger proportion of fluorine than the normal bone on which they are situated. The similarity in the disturbances in ossification caused by a deficiency of vitamin C and by the administration of fluorine suggests a line of approach to this problem.

The Skeleton as a Buffer

The skeleton appears to play the role of a defensive agent against the harmful effects of fluorine. It acts as a ready source of calcium required to detoxicate the fluorine (that probably gets into the blood) and effectively fixes the fluorine so that it is prevented from reaching the softer tissues. When, however, the skeleton is no longer able to take up fluorine rapidly, this element gains access to the soft tissues and causes symptoms associated with the toxic manifestations of fluorine. On this view of the skeleton as a buffer it is possible to understand the period of lag noticed[12, 13] in experimental animals between the commencement of the administration of fluorine and the noticing of symptoms such as loss in weight. It is also possible to explain the sudden increase in the level of urinary excretion of fluorine[14] after an initial period of fluorine

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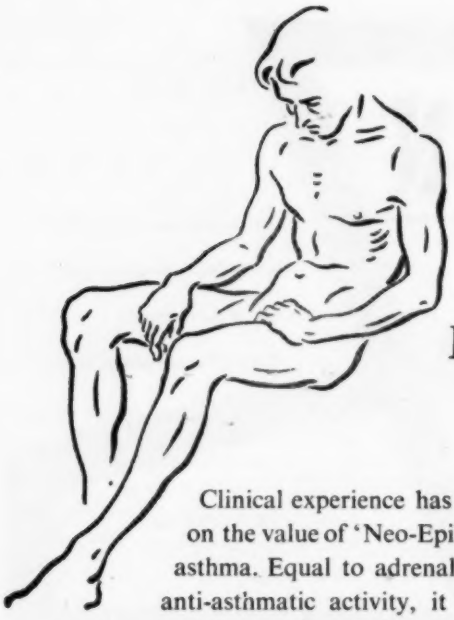

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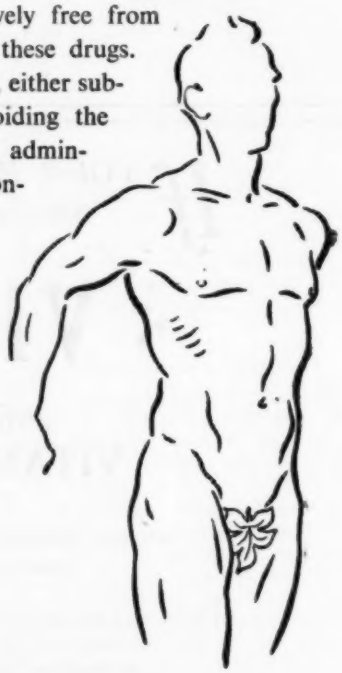

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administration during which the excretion of fluorine is not high. This view also explains the beneficial effects of dietary supplements of calcium in experimental animals which are fed fluorides at levels which cause death or decrease in the rate of growth.

This view of the skeleton as a buffer does not, however, exclude the possibility of the bone system itself being injured in the process. According to Largent[15] deformities and exostoses of various types may exist even if they are not demonstrable under the x-ray.

Antidotal Agents

(1) *Calcium Supplements*—Several methods have been suggested for mitigation of the harmful effects of ingested fluorides. The observations of McClure and Mitchell[15a] that the retention of calcium in the body is reduced by feeding fluorides to experimental animals led to the investigation of calcium supplements as an ameliorative measure. Schulz[4] noticed that the dietary supplements of calcium carbonate mitigated the adverse effects of fluorine on the growth and reproduction of rats. Ranganathan[16, 17] reported that calcium supplements prolonged the period of survival of rats receiving fluorine. Pillay *et al.*[18] were able to counteract the decrease in the rate of growth of fluorine-fed rats by supplements of milk powder, bone meal, eggs or fish. Majumdar *et al.*[5] were able to check the loss of weight of bull calves (receiving one gram of sodium fluoride per day) by administration of calcium phosphate but not by administration of calcium carbonate. The animals receiving calcium carbonate supplements were even worse than those receiving fluorides alone thus pointing out the importance of not losing sight of the necessity to maintain the optimum ratio of calcium to phosphorus in our anxiety to mitigate the symptoms by feeding calcium salts.

The adequacy of calcium and phosphorus (in the physiologically optimum ratio) in the diet merely ensured the availability of the bone-forming materials in the proper proportion to meet the rate of bone-formation as enhanced by the influence of fluorides and thus may prove to be of particular importance in the growing animal, which is saved an undue strain on its skeletal resources of calcium.

The deposition of bone-salt continues to be irregular and atypical since it takes place under the influence of fluorine. It follows, therefore, that dietary supplements of calcium and phosphorus cannot prevent the formation of the osteophytic outgrowths. In fact, Majumdar[5] found that the animals receiving supplements of calcium "were not free from exostoses." Similarly, Marx[17a] records that the administration of parathyroid did not prevent the osteodystrophy in guinea-pigs receiving fluorine. Roholm[6] quotes the work of Rost who came to similar conclusions.

Sooner or later, either as a result of skeleton reaching its limit as an efficient buffer or as a result of the cessation of the formation of new bony tissues, the ingested fluorides would be able to leak through the more vulnerable organs.

Thus, while calcium supplements are valuable in preventing the undue drain on the skeleton and can be expected to postpone the harmful effects of fluorides, they cannot be looked upon as an antidote against continued ingestion of fluorides or against the tendency to form the troublesome osteophytic outgrowths and calcification of the ligaments.

(2) *Vitamin C*—In 1931 Hauck and Steenbock[19] showed that the administration of orange juice influenced favourably the rate of growth and period of survival of rats receiving fluorides. Tauber and Kleiner[20] came to similar conclusions but Ranganathan[17] was unable to notice any favourable effect of vitamin C in his fluorine-fed rats. The observation of Fargo *et al.*[21] that the adverse effects of feeding a rock-phosphate containing fluorides were less noticeable among those swine which had access to green pasture would appear to support the view that vitamin C has a beneficial effect. Pandit *et al.*[22] found marked differences in the severity of the symptoms of fluorosis among the natives of two adjacent villages whose water supplies contained the same amounts of fluorine. Dietary surveys showed that the calcium intake by the people in the two areas did not differ significantly but that the diet in the more severely affected village was poorer in its content of vitamin C. Experiments with monkeys[22] supported this view that the deficiency of vitamin C accentuates the symptoms of fluorosis.

There appears to be some relation between fluorine and vitamin C. Some of the symptoms caused by the deficiency of vitamin C are also provoked by the administration of fluorides to experimental animals[19, 24]. The healing of induced scurvy by the administration of orange juice was hindered if fluorides were administered simultaneously[24]. Biester *et al.*[25] found that the degenerations of the myelin sheath of the spinal cord provoked by a deficiency of vitamin C in the diet of dogs were accentuated by the simultaneous administration of fluorides.

The disturbances that are induced in normal ossification as a result of the deficiency of vitamin C make this subject particularly interesting from our point of view of preventing the undesirable skeletal changes caused by fluorine. Roholm[6] quotes Hojer *et al.* to say that a deficiency of vitamin C causes disappearance of the collagen tissue and increases degenerative amorphous calcification. Mouriguand *et al.*[26] showed that chronic scurvy in guinea-pigs leads to peripheral ossification at the expense of the central bone structure and that characteristic osteophytic outgrowths can be induced in guinea-pigs by feeding a diet deficient in vitamin C. Murray and Kodicek[27] observe that new bone formed at the site of a fracture in guinea-pigs showed much larger thickenings if the animal was on a diet deficient in vitamin C. It is interesting to recall here the observations of Morgareidge and Finn[11] on the

bone deposited at the site of a fracture under the influence of fluorine in the diet.

Since the storage of vitamin C in the liver and adrenals of experimental animals receiving fluorine is not significantly reduced[19, 27, 28] it is suggested that chronic fluorine intoxication may interfere with the mechanism by which vitamin C functions in the body[29, 28]. It may perhaps even be suggested as a working hypothesis that the osteophytic outgrowths noticed to occur under the influence of fluorine are really an indirect effect caused as a result of interference with the utilisation of vitamin C by the body.

(3) *Aluminium salts*—Smith and Leverton[30] noticed that cryolite is not as toxic to rats as an equivalent amount of sodium fluoride. Kempf *et al.*[31] found that aluminium fluoride did not produce mottling of the teeth or other adverse effects. The simultaneous administration of aluminium salts was found to diminish the toxicity of calcium fluoride, natural rock phosphates containing fluorides[32] or of sodium fluoride[33, 34] added to the diets of rats, rabbits, guinea-pigs or bull calves. Aluminium salts were effective in protecting rats and rabbits even against lethal doses of fluorides[35].

The mottling of the incisors of the rats did not proceed beyond the very mild stages when the animals received a supplement of aluminium sulphate[37]. A noteworthy feature about the ameliorative effect of aluminium salts is that they appear to be capable of preventing the formation of exostoses (Majumdar) besides checking the deterioration in the general condition and in the blood picture[5].

The mode of action of aluminium supplements has yet to be elucidated. The fæcal excretion of fluorine is not increased under the influence of aluminium salts but the decreased storage of fluorine in the bones[37] (as compared to the bones of the control animals receiving fluorine without alum-supplements) suggests that the assimilation and metabolism of this toxic element is somehow hindered.

Withdrawal of Fluorine

While the withdrawal of fluorine does not strictly come under the category of antidotal measures, it is desirable to consider whether the conditions induced by fluorine can regress if the fluorine is withdrawn.

It has been so well realised in South India that cattle which have lost condition and developed a dragging gait as a result of living in areas of endemic fluorosis can recover if they are shifted to a place outside the endemic area, that cattle merchants ply a thriving trade buying cattle cheap from endemic areas, improving their condition and selling them at a profit. Pierce[38] showed that sheep which had been losing weight as a result of administration of fluorides improved their condition and gained in weight when the fluorides were withdrawn. Rats receiving fluorine showed a similar ability to gain in weight and grow normally when the fluorine feed was discontinued.

The elimination of fluorine continues even after the fluorine feed is stopped. The elimination is rapid at first and then continues at a slower rate[39]. The fluorine content of the bones similarly falls rapidly at first and more slowly thereafter[40]. The examination of the bones of a man who died of other causes 17 months after he ceased working in a cryolite factory showed (Roholm[6]) that the areas of excessive decalcification tended to undergo a re-organisation.

Summary

The two main symptoms of chronic intoxication by fluorine are (1) mottling of the tooth enamel and (2) skeletal changes such as the formation of " exostoses " on the long bones and " calcification " of the ligaments. Toxic symptoms (such as decreased rate of growth or even death) which occur when higher levels of fluorine are ingested are not so common.

Mottling of the enamel is due to a direct effect of fluorine on the enamel-forming mechanism. Since the enamel-forming cells disappear in later life, this condition is not reversible in human beings. Common dietary supplements do not prevent mottling of the tooth enamel but the administration of aluminium salts appears to have a beneficial effect.

In chronic fluorine intoxication, the skeleton plays the role of a defensive mechanism. It acts as a ready source of calcium needed to detoxicate the fluorine and at the same time immobilises the fluorine by storing it and thus prevents the fluorine from reaching the soft tissues. However, during this process of acting as a " buffer " the skeleton may not escape injury. Deformities and exostoses of various types may occur. Since these skeletal changes can be caused particularly in our country—by water-borne fluorides and since such changes can cause considerable discomfort, it is desirable to shift the emphasis of investigations on methods which could prevent their formation or cause the regression of such changes as have already set in.

The mechanism of formation of these exostoses has yet to be elucidated. Dietary supplements of calcium and phosphorus can protect against the drain of calcium from the skeleton and against the toxic manifestations of fluorine but not against formation of exostoses. On the other hand, the administration of aluminium salts or of vitamin C offer promise in this direction and are, therefore, worthy of a closer study.

In the meanwhile, the reduction of the fluorine-content of water to non-toxic levels and more thorough investigations which would be expected to result in the exclusion of other sources (such as cooking water) of fluorine would appear to be worth pursuing not only because such methods are the best and most reliable methods of preventing the undesirable symptoms, but also because they appear to offer promise of inducing regression of the skeletal manifestations that have already set in.

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CASE NOTES

NEUROFIBROMA OF THE CERVICAL REGION

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Intraspinal tumours are only about one sixth as common as intracranial tumours. According to Elsberg's studies only about 2 per cent of these are neurofibromas and nearly half of them are in the cervical region, and 40 per cent of them had extraspinal extensions. Ewing in 1931 collected 400 spinal cord tumours of which only 37 (0·9 per cent) were neurofibromas.

These neurofibromas arise from the sheaths of the spinal nerves and are encapsulated and circumscribed. The nerves from which they arise can often be traced to one pole of the growth. These tumours can start within the dura or outside the dura. Occasionally, such a tumour may extend along the nerve from inside the dura and come extradurally and then extend through the intravertebral foramen into the extraspinal tissue. Because, they are constricted at the point where they pass through the duramater or at the intravertebral foramen, such tumours are called hour-glass or dumbbell tumours. They are usually single, but occasionally they may be present on several nerve roots especially in cases of generalized neurofibromatosis of von Recklinghausen.

The symptoms of these extramedullary tumours almost invariably develop very slowly and continuously although cases have been described of sudden onset or with decided remissions and exacerbations. These symptoms may be divided into (1) those due to involvement of nervous structures arising at the level of the growth, and (2) those caused by interruption of pathways to and from the subjacent segments.

Better brilliant results cannot be found in surgery than those following successful removal of these tumours and fortunately the majority of these tumours are removable. The symptoms are relieved, the bed-ridden patient walks again, and the paralysed and helpless resumes his former life of activity.

Case Report

Masillamani Pillai, male, 35 years old, was admitted in Christian Medical College Hospital, Vellore, on 31-8-1950 with the following history:

(1) Gradually increasing difficulty in walking for the previous one year, more marked for five months and completely bed-ridden for twenty days.

(2) Increasing weakness of both hands—duration five months.

(3) Retention of urine and constipation for the previous twenty days when he was admitted in another hospital where he was being catheterised.

(4) There was a lump on the left side of the neck, noticed first about five years ago which was gradually increasing in size. There was occasional pain in the swelling, especially on pressure, but he had not noticed any difficulty in swallowing or any other pressure symptoms.

(5) Twenty years ago, when the patient was 15 years old, he had acute pains in the left hand which was radiating from the shoulder down the ulnar aspect of the fore-arm to the little finger. This lasted for a few months and with treatment by indigenous methods the pain got better but there was loss of sensation and weakness of the little finger. This gradually progressed to a typical claw-hand about 15 years ago. During the last one year with the increasing weakness of the left hand, he started to drag his left leg and later the right leg and for the last twenty days his right hand also had become weak. There was much pain in the neck on movement for the last one month.

There was no history of venereal disease.

Family history and enquiry into the various systems did not reveal anything contributory.

On Examination: A fairly nourished individual with no abnormality detected in the heart, lungs or abdomen. B.P. 130/85 mm. of Mg.

On the left side of the neck there was a firm mass about 3" × 2" × 2" underneath the sternocleidomastoid muscle. The skin was not adherent to the mass, which was not fixed to deeper structures. This mass was tender.

Two neurofibromatous nodules, one over the left fore-arm and the other over the right side of the chest were noted.

No deformity of the spine was present. There was tenderness over the 4th, 5th, 6th and 7th cervical spines. All movements of the neck were painful. Rectal examination did not reveal anything abnormal.

Nervous System: Intelligence, memory and speech—good.

Cranial Nerves—No abnormality detected.

Motor: Upper Extremity—Power: Movements of both the shoulder joints moderate; but the power of the muscles of the shoulder joint are very markedly diminished. Movements are diminished at both elbow joints and only very little at the wrist. Grip is extremely poor in both hands, no movements in left little and ring fingers.

Tone is very poor in all the muscles of both upper extremities.

Atrophy is noted in all the small muscles of the left hand and also there was typical claw-hand deformity in that hand.

No fibrillations are seen in the muscles.

Lower Extremity—Complete paralysis of all muscles is present and the muscles are spastic. With slight stimulation there are flexor spasms and often both feet have clonus, with slight tension or in certain positions; no atrophy or fibrillation noted.

Sensory—Pain, touch and temperature slightly diminished below C_5 level. Joint sense and two-point discrimination lost in both lower and upper extremities.

Reflexes—Deep: Biceps, triceps, supinator and pronator are all brisk and equal on both sides. Knee, patellar, and ankle jerks are markedly exaggerated on both sides. Marked patellar and ankle clonus noted on both sides.

Superificial: abdominal and cremasteric completely lost and plantar response was extensor.

The patient has retention of urine.

Laboratory Findings

Blood—Hb.—15 gms; W.B.C.—20,800/c.mm.; poly—86 per cent, eos—4 per cent, lymph—9 per cent and mono—1 per cent. Kahn test—Negative.

Urine—Reaction—alkaline, albumin—two plus, sugar—negative, pus cells—two plus, R. B. C.—two plus.

C. S. F.—Kahn—Negative, cells—4/cm., chlorides—719 mgm. per cent, protein—260 mgms. per cent and sugar—71 mgm. per cent.

Therapeutic Procedure

1. Graduated manometric study: showed complete block.

2. X-ray cervical spine: A.P. and lateral views: Destructive defect of the pedicles and laminæ of C_3, C_4 and C_5 on the left side. Left side of the body of C_3 and C_4 were also involved. The intervertebral space between C_3, C_4 and C_4 and C_5 were narrowed.

Diagnostic Procedure

Operation was done in two stages:

First Stage—Removal of the extradural part of the tumour on 7-9-1950. Laminæ and spines of C_4, C_5, C_6 and

C_7 were exposed. There was complete destruction of pedicle and laminæ of C_5, C_6, and C_7 on the left side. A pinkish hard tumour was pressing the cord markedly, and there was no pulsation of the cord below the tumour. The tumour had destroyed the part of the left side of the bodies of C_6 and C_7 and was extending anteriorly extraspinally. The tumour was removed as much as was possible releasing the pressure on the cord, which immediately started to pulsate. Tibial graft was placed on both sides from C_5 to T_1 and wired in place. The wound was closed in layers.

Second Stage—Removal of the tumour from the neck. This was an easy procedure as the tumour was not adherent to any structure. The portions of tumour near the vertebral bodies were carefully removed and wound closed in layers.

COURSE IN HOSPITAL: The patient made an uneventful recovery. Wounds healed by primary intention. After the removal of the stitches the patient was given a plaster jacket including the neck. The recovery of nervous function started the day after the operation.

On the 3rd day, the patient started feeling stronger in the limbs with complete disappearance of flexor spasm, and clonus, and plantar response was flexor.

On the 7th day, he got his bladder function back.

From the 12th day onwards there were no positive neurological findings except the wasting of the small muscles of the left hand.

The patient was discharged on 22-10-1950, and he walked out of the hospital.

The plaster jacket was removed on 26-10-1950, and re-examination of the nervous system showed no positive neurological findings except that the small muscle of the left hand was wasting though, the claw-hand was much better and the power was fair and he could use that hand.

PATHOLOGY REPORT: *Macroscopic*—The tumour at the first operation weighed 20 gms. and measured 3 cm. × 3 cm. × 3 cm. It was encapsulated, hard and brownish in colour. The tumour cut with resistance and the cut surface appeared dull-white with yellow mottling and areas of hæmorrhage. The tumour in the neck was 45 gms. in weight and the colour and consistency and cut surface were similar to the intraspinal tumour.

Microscopic appearance was typical of perineural fibroblastoma. The pallisading of the nuclei, which was considered as pathognomonic of these tumours were seen. In addition to the actual pallisade arrangement of nuclei they tended to be arranged in linear pattern. The nuclei were elongated and sometimes curved. There were occasional degenerating areas. Silver stain showed parallel fibres of even calibre. This picture was seen in both portions of the tumour.

COMMENTS

The tumour was present over twenty years and most of the symptoms were due to motor and not to sensory deficits. So the tumour evidently arose from the motor root. The development of claw-hand showed that more than one nerve root was involved due to the pressure of the tumour. Widespread destruction of the laminæ, pedicles and body of the vertebræ by the benign growth were seen.

TRANSPLANTATION OF URETERS INTO THE PELVIC COLON

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In this operation, the flow of urine is diverted from the ureters into the pelvic colon, from where it can be excreted per rectum when the sphincter control for the liquid contents of the lower bowel is established. Two cases of inoperable vesicovaginal fistula where this operation was performed are presented here. Other conditions for which this can be employed are ectopia vesicæ, congenital defects associated with incontinence, injury above the triangular ligament, cases of carcinoma of the bladder before total cystectomy, incurable cystitis, some cases of contracted bladder due to tuberculous cystitis, and accidental injuries in pelvic operations.

Both the cases mentioned below, were unsuitable for local repair. When plastic repair is impossible transplantation of the ureters is the only operation which can relieve the urinary incontinence and the suffering which accompanies this condition.

Pre-operative management—It should be noted that this is not an emergency operation. Most of the patients are run down, due to infection, loss of sleep and psychological causes. The general health is improved with diet, vitamins, liver extract, and blood transfusion, whenever necessary.

Renal function tests should be carried out. In some cases, pyelography may be indicated. Attention should be given to the urinary infection. If salpingitis, erosion and tears of the cervix, or pelvic inflammation, is present, this should be properly treated before undertaking the operation. Chemotherapy reduces the risk of post-operative infection, sulphasuxidine or pthalylsulphathiazole may be used for this purpose. These can be given for several days before and after the operation. The dose employed is 0·25 gm. per Kgm. of body weight daily for 5 days before the day of the operation.

Operative Technique—Modified Coffy II technique is often preferred. The one-stage operation is considered better than the former operation done in two stages. Recent experiences of Lakhani (1948) and Rao (1949) also confirm this. During operation the following conditions are fulfilled:—

(*i*) Provision of good drainage of urine from the kidney to the bowel. This is achieved by avoidance of ureteral compression, angulation and stomal œdema.

(*ii*) Absence of tension at the site of anastomosis.

(*iii*) Preservation of the blood supply of the ureter to prevent necrosis of the ureter and peritonitis.

(*iv*) Very high Trendelenburg position is essential during the operation and a good self-retaining retractor is also necessary.

(*v*) In order to maintain satisfactory urinary output glucose saline infusions are essential.

(*vi*) The actual implantation of the ureters in the colon is done after Witzel's operation. This prevents ascending infection and the oblique implantation produces a valvular effect.

The site of election in the pelvic colon for the implantation is just below the pelvic brim. The right ureter is transplanted first and the left at a higher level.

Some urologists recommend the sodium-sulphate injections (4·3 per cent) by drip method as a routine after the operation.

Complications—Complications of this operation are: anuria or oliguira, lung complications, peritonitis, pyelitis and ascending renal infections, extra-peritoneal inflammation, rectal irritation. In both of the cases under review the author had none of these complications.

Post-operative Care—The kidney function is watched. Plenty of fluids and urinary antiseptics are essential. The total intake both by mouth and injections should be about 150 oz. per day. If the quantity of urine is less than 600 c.c. the patient is given a pint of 4·3 per cent solution of sodium sulphate. Some urologists advocate the use of this drug as a routine in the post-operative period. The urinary output is measured every day. The catheters come out of themselves between the 8th and the 12th day after the operation. The patient is put on non-residual diet from the 2nd to the 10th day.

The mortality in Rao's series (1949) was about 6 per cent, while Dr. Lakhani, (who has reported about 7 cases in case of carcinoma of the bladder), reports about 10 to 15 per cent mortality in his series. Both the cases presented here were cured within 3 to 4 weeks after the operation. They are reported to be fit, and have good urinary control.

Case Reports

Case 1—A 25 year old hindu female was admitted on 12-5-50 with a vesicovaginal fistula. She was a primipara. About 1-1/2 months before admission, she was delivered after 3 days of labour with the aid of forceps.

Physical Signs: The patient looked anxious, worried and anæmic. She could not walk as a result of pelvic cellulitis and mild phlebitis.

Vaginal Examination: The anterior vaginal wall had sloughed out completely and the fingers went through the fistula and touched the fundus of the bladder. There was thick scarring tissue surrounding the fistula. The cervix of the uterus could not be felt easily but it was adherent to the posterior vaginal wall embedded in the cicatix.

Speculum Examination—A large fistula was seen in the anterior vaginal wall. The cervix was seen with great difficulty in the posterior fornix, and it bled on touch. The ureteral openings were searched for and found in the vault of the vagina. These were verified by anæsthetising the patient and passing ureteral catheters.

Pre-operative Treatment: The patient was prepared with regular vaginal douching, tonic mixtures, calcium lactate by mouth. One injection of milk 5 c.c. was given for the pelvic cellulitis. They day before the operation the ureteral catheters were passed as described above.

Operation: The operation was performed on 26-5-1950. A modified Coffy's II operation was done. Plasma was kept ready in case it became necessary. Glucose-saline was started. A midline incision was made. A very high Trendelenberg position was used. The intestines were packed away. There was difficulty in locating the ureters at the pelvic brim, because of the presence of big pelvic blood-vessels. But after a little time the left ureter was found out and dissected out for about 1½". A No. 2 fine rubber catheter was put in and tied in. A flatus tube was inserted through the rectum upto the pelvic brim. The right ureter was similarly dealt with. The flatus tube was drawn out by an assistant by which the rubber catheters were pulled out together with the ureteral ends which lay on the pelvic colon. The ureters together with the rubber tube inside were stitched in the pelvic colon so as to lie in the submucous tissue obliquely as in Witzel's operation. The operation took 2½ hours to complete. The patient was returned to bed and glucose-saline was administered.

Postoperative Course: The flatus tube was maintained in the rectum. Penicillin and soluseptasine were administered parenterally and sulphadiazine was given by mouth. Powders consisting of urotropin, potasium citras and sodium sulphate were given by mouth to act as diuretics and laxatives. The left catheter came out on the 8th day and the right on the 11th day. A rise of temperature on the 12th day was controlled with injections of pyelopurin and glucose.

She developed good control of the rectal sphincter for urine, and was discharged 5-7-1950.

Case 2—A 28 year old hindu female was admitted on 1-8-1950 with a diagnosis of vesicovaginal fistula.

History: The patient had developed vesicovaginal fistula after a forceps delivery two year before. She was operated three times but without any effect.

Vaginal Examination: There was a big fistula which admitted two fingers.

The patient was prepared with regular vaginal douching and glucose saline and calcium injections given for fifteen days.

Operation: She was operated on 17-8-50. Exactly the same technique was adopted as in Case 1. The patient had an uneventful recovery and was discharged cured on 13-9-50.

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DETOXICATION OF ARGEMONE OIL ADULTERATING MUSTARD OIL

After Lal and his co-workers[1] had finally established that epidemic dropsy was caused by the ingestion of mustard oil adulterated with argemone oil, the practical effect of this discovery became evident by the non-appearance of the epidemic for several years following. But this state of improvement in the epidemic dropsy situation suffered a considerable set-back in 1949 when a large number of samples of mustard oil examined by Seal at the All-India Institute of Hygiene and Public Health, Calcutta, in the month of June of that year were found more or less adulterated with argemone oil. Following this, several large consignments of mustard oil were found similarly adulterated, by the Health Department of the Calcutta Corporation. The epidemic reappeared in the city and the events that followed are perhaps still fresh in the memory of the public as much as of the practising physicians.

Since the quantities involved in the prosecutions were considerable on the face of shortage of this essential article of food in this part of the country and the samples available in the market were generally under suspicion, the question of a simple method of detoxication was raised from many quarters. The parties who were, however, mostly interested in the discovery of such a process were the oil merchants and the dealers who had staked large capitals in that business. But to allow the adulteration to go on unchecked and then adopt detoxication would be equivalent to burning the wick out at both ends, a principle logically unsound. This situation arose due to the absence of rigid legal procedures and measures needed in the country to prevent admixture of mustard seeds with argemone seeds in the fields or before the milling stage. Actually, the Health Department of the Calcutta Corporation had to face considerable difficulties in prosecuting genuine cases of adulteration which severely endangered the public life.

It is known that argemone grows wild in certain seasons in U. P., Bihar and also in various other parts of the country and the adulteration is generally done to increase the yield of oil, with or without the knowledge of its toxicity. Thus, legislation prohibiting the cultivation of argemone and enforcing the compulsory weeding out of the plants when growing wild in and around the mustard fields, through the Agricultural Department would have been the correct and simple preventive measure. In addition, heavy penalties should have been levied on the millowners for storing and using argemone-adulterated seeds in their mills. As a matter of fact, with a little training the seeds can be easily detected and removed by mechanically driven Taylor's standard seives of mesh 10 to 11 to the inch (opening less than 0·065 inch).

As regards the other aspect of the problem, namely, the process of detoxication mention may be made of the work of Lal *et al.*[2] (1940) who claimed to have reduced the toxic substance in the mustard oil to one-tenth (from 5 per cent to less than ½ per cent) by exposing the adulterated oil to sunlight and air for about 50 hours or more, as well as by ultraviolet irradiation in the presence of air. But it is a question whether it would be possible to detoxicate adulterated mustard oil in bulk by these means. In a subsequent work Mukherji, Lal and Mathur[3] (1941) removed the toxic substance of the oil or at least removed the alkaloidal principle responsible for the chemical test, by passing dry HCl gas through an ethereal solution of the adulterated sample. Though the residual oil was non-toxic to human beings it had lost its edibility, and the process was neither simple nor economic. The observation of Chopra *et al.*[4] (1939) that argemone oil heated to 240°C. lost its toxicity to laboratory animals was not confirmed by human feeding experiment nor was it supported by other workers.

It is apparent from the above findings that the toxic principles or rather the one that is responsible for the nitric acid or ferric chloride test contained in the argemone oil could easily be destroyed or modified by oxidation or hydrolysis. In this respect the most effective chemical processes are certain inorganic acids like HCl, HNO_3 etc., but the edibility of the oil is completely lost. Of the other agents tried independently by Roy[5] (1950) and Seal[6] (1950) namely hydrogen peroxide, glacial acetic acid, trichloracetic acid, phosphoric acid and absorbents like Kieselguhr, calcium phosphate, potassium hydrogen phosphates, phosphoric acid has been found by both of them to be the most successful agent for detoxication so far as the test to the reacting substance with nitric acid and ferric chloride reagents and the edibility of the oil are concerned. It seems that the success of the procedure, as made out by Seal, depended upon using anhydrous acid for detoxication and water-free compound like calcium or sodium carbonate (anhydrous) as the neutralising agent. But in view of the fact that the final proof of detoxication should be based on actual human

[1] Lal *et al.*—*Indian J. M. Research*, **25**: 239, 1937; *Ibid*, **27**: 207, 1939; *Ibid*, **29**: 157 & 167, 1941.

[2] Lal *et al.*—*Indian J. M. Research*, **28**: 163-196, 1940.

[3] Mukherji, Lal and Mathur—*Ibid*, **29**: 361, 1941.

[4] Chopra *et al.*—*Indian M. Gaz.*, **74**: 193, 1939.

[5] Roy, A. C.—*Curr. Sci.*, **19**: 91, 1950.

[6] Seal, S. C.—Lecture demonstration in the symposium on Epidemic Dropsy held on 8-3-50 under the auspices of the Calcutta Branch, I.M.A., 1950.

feeding experiment, Seal subscribed to the view that it would be premature and even dangerous to publish the process at this stage for reasons which will perhaps be obvious from the following discussions.

Although the alkaloidal substance isolated from argemone oil by Mukherji *et al.* (*loc. cit.*) accounted for the physical and chemical tests, described either by these workers or by Sarker, the residue left after removal of c.f.b. (reacting substance) did not produce epidemic dropsy in man nor did c.f.b. itself or when recombined with the residue. They, therefore, concluded that the toxic material in the oil had either been broken down or altered during the process of isolation. The empirical formula as given by these workers was $C_{19}H_{15}O_4N$ and comparison with berberine and protopine showed that they were closely allied substances. Four years later Sarker *et al.*[7] (1945) gave nearly the same composition for the orange-red crystals precipitated in ferric chloride test, according to which the formula for the base should be $C_{19}H_{14}O_4N$. Strangely enough, subsequently criticising the formula proposed by Mukherji *et al.*[3], Sarkar[8] (1948) identified the base (c.f.b.) with dihydrosanguinarine having the empirical formula of $C_{20}H_{15}O_4N$ and constituting about 87 per cent of the alkaloidal principles thrown out as precipitate by them. Of the remaining part only 5 per cent was accounted for by him as sanguinarine, and he claimed to have obtained 100 per cent fatal toxic effect in albino rats by giving very heavy doses of sanguinarine hydrochloride equivalent to 9 c.c. of pure argemone oil (*i.e.*, 90 c.c. of 10 per cent or 180 c.c. of 5 per cent adulterated oil commonly found in the market) per rat and came to the conclusion that this substance was the toxic component of argemone oil. It may be mentioned here that according to Henry[9] (1924), sanguinarine ($C_{20}H_{15}O_4N.H_2O$) is the principal alkaloid of *sanguinarine canadensis* (blood root) and has also been isolated from *Bacconia cordata, chelidonium majus* and *stylophorus diphylum*. The three alkaloidal contents of *Argemone mexicana* seeds, so far discovered, are protopine, berberine and argemonine; the last one, described as impure protopine, may be the same identified as sanguinarine by Sarkar. But neither human nor animal experiments have yet been reported with sanguinarine as such and the pharmacological properties assigned to it by Henry in his 1924 edition of "Plant Alkaloids," properties that did not resemble symptoms of epidemic dropsy, have been withdrawn from the 1939 edition on the ground that the separation of sanguinarine from protopine and especially chelerythrine was difficult.

From the above results it cannot be concluded that the reacting substance, which even if accepted as a mixture of dihydrosanguinarine, sanguinarine and some other substance, represents the true toxic substance or its derivative in the argemone oil, that causes epidemic dropsy, even though it may qualitatively and quantitatively indicate the admixture of argemone oil in mustard oil or any other oil. On the same ground the argument put forward by Roy[10] that since the residue left after removal of the reacting substance gave negative chemical tests and also proved non-toxic to human beings, the residual oil left after phosphoric acid treatment giving negative chemical tests should also be similarly non-toxic, is untenable, because while it is possible or even probable that a precursor of the reacting substance in the argemone oil may be the toxic substance it does not rule out the possibility of an entirely different substance being the toxic principle, particularly when Sarker failed to account for 8 per cent of the alkaloids of argemone oil. Even if Sarkar's work were confirmed it could not be said that the causal toxic substance of epidemic dropsy was not included in this remaining 8 per cent. Besides, he makes no attempt to reconcile these findings with his previous results, whereby the composition of his orange-red crystals approximated to the formula proposed by Mukherji, Lal and Mathur[3] (1941). Unless these points are satisfactorily settled it would be incorrect to assume that any or all processes which destroy or remove the reacting substance must necessarily destroy or remove the toxic substance if the latter happens to be different from the precursor of the reacting substance. This hypothesis finds further support in the recent work carried out in this connection by Mitra[11] (1951) who states: "Though the alkaloids sanguinarine and dihydrosanguinarine had been removed from the argemone oil, it has even then produced its effect though in a less marked manner. It may be due to argemone oil itself or some factor or factors other than sanguinarine and dihydrosanguinarine."

Thus phosphoric acid treatment of mustard oil adulterated with argemone oil, as suggested by Roy as well as by Seal would be an important contribution towards detoxication of adulterated oil, provided it would be proved by human experiment that the treated oil is non-injurious and has also retained all the nutritive and edible properties as that of the mustard oil and that the process would be economically feasible on a commercial scale. Besides, the identification of the toxic substance in argemone oil has not been completely established and as such the process of detoxication without the complete knowledge of the toxic principle would necessarily remain incomplete. Further work is, therefore, necessary and until the points are clarified caution is necessary before declaring any process safe enough for public use, because interested people may take advantage of the test and a sense of false security may needlessly endanger many lives.

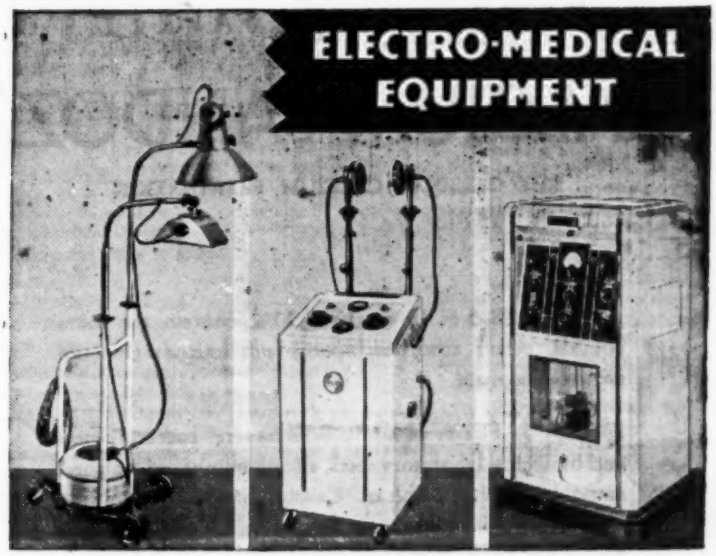
[7] SARKER AND RAHAMAN—*Curr. Sci.* 14: 196 & 201, 1945.

[8] SARKAR, S. N.—*Nature*, 168: 265, 1948.

[9] HENRY, T. D.—The Plant Alkaloids, 2nd Edition, 1924 p. 208, 219 & 313.

[10] ROY, A. C.—Personal Communication.

[11] MITRA, H. N.—*J. Indian M. A.*, 20: 315-16, 1951.



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CURRENT MEDICAL LITERATURE

Lethal Effect of Morphine in Chronic Cor Pulmonale

Roussak (*Lancet*, 1: 1156, 1951) writes that morphine is deadly in heart failure secondary to chronic lung disease. Six patients are described who illustrated this.

Nikethamide, given intravenously, is valuable in rescuing the patient if the ill-effects are noticed in time.

Bursitis

Stone (*Illinois M. J.*, 98: 335, 1950) in reporting on the observations of nineteen cases of bursitis observes: There were eleven females and eight males. The average age in the females was fifty-four years and in the males, fifty-six years. All of the cases were unilateral, fourteen on the right side and five on the left. The subdeltoid bursa was involved in sixteen instances and the acromial bursa in three.

The x-ray findings were positive in 16 cases and were negative in 3 cases. The positive rœntgen findings in 16 cases showed an irregular ring or area of calcification in either the subdeltoid or acromial bursæ.

There are three methods of therapy of bursitis. They are: (1) x-ray irradiation of the calcified ring or shadow; (2) surgical removal; (3) injection of a local anæsthetic (3 to 5 c.c.) into the calcified ring or shadow. This is best done under fluoroscopy. All of our 19 cases received 4 or 5 x-ray irradiations in a period of 2 to 3 weeks. All have made a complete and permanent recovery. After each x-ray treatment there was an exacerbation of severe additional pain in the involved areas. Two weeks after the last x-ray exposure the pain vanished and has not returned to date.

Nineteen cases of chronic subdeltoid and acromial bursitis are herewith reported. All of them are of the chronic type. Sixteen showed positive evidence of calcific deposits or shadows in the involved bursæ either in the right or left upper extremities. All of them made an excellent and permanent recovery with four or five x-ray irradiations. In three cases showing no positive x-ray findings a diagnosis of bursitis was made because of the severe pain over the subdeltoid and acromial bursal regions. X-ray irradiations produced a cure in these three cases. These nineteen cases having severe pain in either the arms or shoulders makes it necessary, in the opinion of the author, to consider it in the differential diagnoses of various syndromes in the shoulder and arm. It also should be considered in the differential diagnosis of cervical rib, affection of the scalenus anticus muscle, various and sundry joint problems such as arthritis, fractures, dislocations and neoplasms.

Cortisone in Chronic Regional Enteritis and Chronic Ulcerative Colitis

Machella and Hallan (*Am. J. M. Sc.*, 221: 501, 1951) write that the purpose of this report is to present observations made when cortisone was administered to patients with chronic regional enteritis or chronic ulcerative colitis. It was thought that the compound might influence the course of the disease because of the occasional association of arthritis and of erythema nodosum.

A trial of cortisone therapy would appear to be worthwhile in certain cases of regional enteritis or ulcerative colitis, particularly in critically ill patients when the situation is desperate and a temporary period of improvement is desired in order to permit sufficient time for the effective use of other therapeutic measures.

The use of the compound does not promise to cure the disease, as a relapse may well occur following cessation of its administration.

Vitamin B Deficiencies after Gastric Operations

Welbourne (*Lancet*, 1: 939, 1951) writes that vitamin B deficiencies are relatively common after gastric operations and have been found in 10 per cent of subtotal gastrectomies one or more years after operation.

Hyporiboflavinosis and incipient peripheral neuritis, due to aneurine deficiency, are the commonest syndromes. Less common conditions are established peripheral neuritis, Wernicke's encephalopathy and other disturbances of consciousness, and pellagra.

In most cases the diets contain sufficient vitamin B. Achlorhydria, which encourages destruction of aneurine and riboflavine and may allow diversion of vitamins to intestinal bacteria, is possibly the chief cause of the deficiencies. Vomiting and intestinal hurry may be contributory factors.

After gastric operations foods rich in vitamin B should be advised. The deficiency states can be easily corrected in their early stages.

Management of Burns

Cram (*Lancet*, 1: 263, 1951) writes that the following simple regime has given good results:

First-aid—The admonitions against applying any kind of grease to burns which may later be treated with tannic acid has resulted in a swingover to no first-aid treatment at all. But it must be borne in mind that the burning agent destroys most of the pathogenic bacteria, and first-aid treatment can minimise infection. Burns on easily exposed areas should have a sterile or clean covering. The cloths should not be removed, even from the burned area—the manipulations required may increase the shock and add injury to the burn. When more than 20 per cent of the body surface is burned in an adult, or 10 per cent in a child, shock is inevitable and early intravenous therapy indicated. If possible, a plasma drip should be set up as a first-aid measure in every case of extensive burns when the hospital for the definitive treatment is more than a few minutes away. An adequate dose of morphine given intravenously allays pain and fear and therefore decreases shock, and when it is given by this route there is no danger of a delayed effect.

General heating of the body is to be avoided, because, as Blalock has shown, the resulting vasodilatation counteracts the reflex vasoconstriction which reduces the discrepancy between the normal vascular capacity and the decreased blood volume. Only enough heat to make the patient comfortable should be applied until the plasma drip is started. The heating of patients to the point where they are actually sweating is particularly deplorable, since the blood volume is further reduced by dehydration.

Treatment in Hospital.—Only if the shock is mild, or when considerable improvement has taken place in the patient's condition, is it permissible to remove the clothes and dress the burns under aseptic conditions.

Local Treatment.—Under adequate sedation, such as intravenous procaine, intravenous morphine, or general anæsthesia, according to the co-operation and psychology of the patient, the burns are exposed for the first time in the clean operating theatre. The foreign bodies, such as cloths, ashes, and loose blistered bits of skin, are thoroughly and gently washed off with cetrimide. There must be no scrubbing, which may injure the thin remaining epithelium, converting a second-degree burn into a third-degree burn without producing any benefit. The prepared area of skin must be kept draped off from the rest of the body as carefully as for an elective operation. Penicillin emulsion or a similar preparation is smeared over the area, which is next covered with a single layer of 'Jelonet' or wide-meshed soft-paraffin gauze. Several layers of gauze 8 in. square are next laid over the burns and firmly bandaged with sterile gauze, or preferably elastic, bandages. It is important that burns around joints should be immobilised in the position of function with plaster slabs. This is particularly important in burns involving the hand. With these burns the fingers must be dressed separately and the slabs either cut separately for each finger or indented so as to keep the fingers apart. Elevation is important in the after-treatment of any extremity burn.

The immediate excision of third-degree burns is an attractive idea, but it is not always possible to differentiate accurately enough between second and third degree, and it had been found that in the absence of infection the area of complete destruction of skin always looks smaller at the second than it did at the first dressing.

The second dressing is done after 7-10 days under the same conditions as the first and with similar aseptic precautions: split-skin grafts are applied at this stage if the areas of third-degree burn are well defined. Weekly dressings are done in the operating-theatre until the burn is completely epithelised.

Treatment of Shock.—The principle of whole-blood therapy for burns is accepted, but its difficulties and dangers make it impracticable for general use.

On the other hand, reconstituted plasma contains too little sodium chloride. Therefore, sodium chloride should be added to the plasma. But there is a danger of salt retention if the fluid intake is insufficient. For this reason the urinary chloride should be repeatedly estimated.

Retinal Changes in Coarctation of the Aorta

Granstrom (*Brit. J. Ophth.*, 35: 143, 1951) writes:

The retinal picture in coarctation of the aorta is often somewhat characteristic, the principal feature being pronounced tortuosity of the arteries.

Tortuosity of the retinal arteries is thus mentioned in many of the cases reported in the literature. In no instance, however, has it been stressed that the retinal changes in this disease are in any way characteristic.

Between 1947 and 1950, forty undoubted cases of coarctation of the aorta were sent from the cardiological department for ophthalmological examination, and it was soon noticed that tortuosity of the arteries was characteristic of many such cases. It was often strikingly regular, and, in some less marked cases, it was confined to certain arterial branches, whereas in others it could be seen in the majority of the branches. Initially, the tortuosity appeared to be associated with regular, undulating changes in the width of the artery, and with regular changes in the light reflexes. The examination was always made with Gullstrand's binocular opthalmoscope, in which a stereoscopic picture of the fundus is consistently obtained. This soon revealed that these changes in the arterial width and in the light reflexes were, in all probability only apparent. It was possible to see clearly how the arteries were often twisted in the shape of a corkscrew, and were, in some places, somewhat embedded in the retinal tissue, whereas other sections of them lay more superficially. This explains the regular changes in the light reflexes and the apparent variations in calibre. (In looking at a spiral metal thread, the changes in the light reflexes are noticed immediately and also—particularly under certain conditions of illumination—apparent variations in width).

More or less pronounced tortuosity of the retinal vessels may occur in normal cases, although it is then, as a rule, mainly localized to one plane and is infrequently found to be in the shape of a corkscrew. Moreover, in normal cases, it is often present in both the arteries and the veins. In coarctation of the aorta, on the other hand, the veins are seldom tortuous.

In a few cases there also appeared to be a generalized narrowing of the arteries. Local arterial constrictions—which are often found in essential hypertension—were, on the other hand, usually lacking. Slight changes of this kind or slight arteriovenous crossing signs were present only in a few instances. In no case could be found hæmorrhages, white patches, or other retinal changes occurring in severe hypertensive retinopathy. In this disorder, tortuosity of the arteries, even in the form of a corkscrew, is sometimes found, particularly in the macular region. In such cases, however, other typical arterial changes are always present concurrently. In coarctation of the aorta, on the other hand, the arteries have an essentially normal appearance, with the exception of the tortuosity. Moreover, the tortuosity is not especially confined to the macular region.

It, therefore, appears evident that the retinal picture in coarctation of the aorta differs distinctly from that usually found in other forms of hypertension.

(1) Corkscrew-shaped tortuosity of the arteries is common in the former disease, particularly in patients over the age of 20 or 25.

(2) The retinal changes characteristic of hypertension of other origin are wholly or partly lacking in coarctation of the aorta.

Tortuosity of the arteries, often in corkscrew form, is common in the entire upper half of the body in coarctation of the aorta, becoming more pronounced with the years. Tortuosity of the retinal arteries is also more frequent with advancing age. It, therefore, appears probable that the tortuosity of the retinal arteries is only a partial symptom of generalized arterial tortuosity, which is a result of the abnormal mechanical conditions.

Hypertension in the upper half of the body is also, presumably, mainly due to mechanical factors. Some authors have assumed that it is caused by a decrease in the blood supply to the kidneys, but other writers have put forward strong arguments against this hypothesis. In coarctation of the aorta, the absence of marked retinal changes, other than tortuosity of the arteries, is, a strong indication that hypertension in this disease is, of an entirely different nature from that found in other hypertensive disorders. In the latter, the condition of the finer arterial branches is probably of prime importance, both for the occurrence of hypertension and for

the possible retinal changes. Conversely, in coarctation of the aorta, the arterial changes (tortuosity) are of a more secondary nature. It is probable that both the tortuosity of the arteries and the hypertension are mechanically conditioned by the increased resistance. The pathogenesis of other hypertensive disorders is certainly considerably more complicated.

Ocular Disturbances during Malnutrition

Petzetakis (*Press Med.*, **58**: 1084, 1950, Ref. *Am. J Ophth.*, **34**: 138, 1951) writes:

These observations were made during the years of hunger in Greece from 1941 to 1944. The word trophopenic was coined to describe the nutritional disturbance which follows famine and is characterized by œdema and by motor, sensory and trophic disturbances. In the eye the chief symptoms consist of œdema of the lids and conjunctiva, corneal anæsthesia, diminshed pupillary reflex, dryness of the globe and changes in the cornea. Diplopia and xerophthalmia occurred rarely, as did hemeralopia. The eye ground was normal except for anæmia, and visual disturbances were absent or slight. The lesions are degenerative, not inflammatory and are due only in part to lack of vitamins: hypoglycæmia, hypoproteinæmia and hypolipæmia are present. The eye is but one of the tissues affected by the disturbances in the peripheral and central nervous system.

A diet rich in protein, fat and vitamins A, B and C brings about a rapid cure in most patients although some die in spite of treatment and in these the eye changes persist until the end. Beer is almost a specific therapeutic agent; opacities clear, keratomalacia heals and nervous and intestinal disturbances abate soon after drinking it.

In the 331 cases observed autopsy was carried out in 11, with histologic studies of the eye. Oedema of the fibers and vacuolar degeneration were characteristic of trophopenic keratitis.

Eye Findings in Juvenile Diabetics

Kumm and others (*Klin. Monatsbl. f. Augenh*, **116**: 482, 1950, *Ref. Am. J. Ophth.*, **34**: 161, 1951) write:

Among 160 juvenile diabetics 33 exhibited retinal disturbances. Seventeen patients had punctate hæmorrhages, 7 had waxy exudates and 9 had both. The retinal disturbances are more often seen in light than in severe diabetes. The duration of the disease was less than 10 years in 12 patients and of only a few months in two. The blood pressure in juvenile diabetics is usually lower than normal. The hypertension in old diabetics seems, therefore, to be a secondary complication. Aimless insulin-therapy, frequent changes in the dosage and abstinence lead to capillary damage and to vascular retinal lesions.

In 11 among 176 juvenile diabetics bilateral lens opacities were found which were ascribed to diabetes. Three patients had the so-called snowstorm cataract. No relation could be detected between cataract formation and blood sugar level and insulin therapy did not resolve opacities.

The authors found transitory myopia in 66 cases. This high incidence might be ascribed to the fact that patients during the war received insufficient or no insuline therapy. Disturbed mineral balance in diabetics causes osmotic pressure changes in the lens leading to increase of volume of the lens fibres and to alteration of curvature of the lens fibres and to alteration of curvature of the lens surface and to myopia. The author observed radial figuration in the retinal reflex during skiascopy of 60 per cent of the myopes and in 20 per cent of the transitory hyperopes, when fully corrected. This may be of diagnostic significance. Slit lamp examination has not revealed any alteration in the lens fibers.

Whooping Cough

Banks (*Brit. M. J.*, **1**: 680, 1951) in dealing with treatment of whooping cough writes:

Treatment of Symptoms.—There is no drug treatment which consistently produces dramatic improvement in whooping cough. Only the course of the disease can be modified and serious complications prevented. This is more easily done in summer than in winter, when so many adverse factors such as cold damp weather, upper respiratory infections, influenza, measles, or pneumonia interfere with the action of a remedy, and lead to a return of coughing and vomiting.

Antispasmodics and Sedatives.—Bromoform drops, one for each year of age up to 6, or elixir bromoformi, ½ to 2 dr. (1·75 to 7 ml.), or sodium bromide, codeine, or phenobarbitone are commonly used as sedatives. Ephedrine, ¼ to ½ gr. (16 to 32 mgm.) twice daily for an infant, and tincture of stramonium or of belladonna, 5 to 15 min. (0·3 to 0·9 ml.), have been much used in an attempt to relax spasm of the bronchial muscle. In small doses these drugs may do little more than irritate the stomach and may actually increase vomiting. Toxic doses will damp down the cough, but this degree of sedation is harmful. By reducing the force of the paroxysms these drugs may encourage the retention of mucus and pulmonary collapse. It is difficult to steer a middle course, and before prescribing such drugs each case should be considered on its merits. Phenobarbitone is probably as good a sedative as any, but it should not be prescribed in the presence of persistent cyanosis, or signs of pulmonary collapse or bronchopneumonia. In infants it may be given in doses of ¼ gr. (8 mgm.) thrice daily. Elixir of luminal (¼ gr. in one teaspoonful) is a convenient preparation. Atropine methylnitrate ("eumydrin"), which is used for dilating the pyloric sphincter in pyloric stenosis, can also be given as a sedative in 0.6 per cent alcoholic solution, four to eight drops four-hourly. It has the advantage of not increasing vomiting and may sometimes relieve it.

Expectorants.—Ipecacuanha, ammonium carbonate, potassium iodide, and large doses of alkaline mixture for the older patients are all employed in what is often a vain endeavour to thin or dissolve the bronchial mucus. These drugs increase salivation and may induce vomiting. They are occasionally useful from the fourth week onwards, when the mucus is very viscid, but should not be prescribed as a routine or for long periods. They are hardly at all used in hospital.

The Decompression Chamber.—This treatment is the equivalent of an aeroplane flight at 12,000 feet for 45 minutes. It is suitable only for uncomplicated cases after the third week of the disease as an aid to clearing up severe paroxysms and stopping vomiting. It is contraindicated in pyrexial or pneumonic cases or those with cyanosis, but not in apyrexial cases with limited pulmonary collapse.

Specific Treatment:—Pertussis Vaccines: There is no convincing evidence that vaccines have any appreciable effect in the treatment of the ordinary cases of whooping cough in a child.

Chloramphenicol:—This is probably the best specific drug for whooping cough, but its action is rarely dramatic. It is a very expensive drug and should not be wasted on mild or late cases or where an inadequate course is likely to be

given. It should be given preferably before the twelfth day of the disease and only in cases which appear to be developing severe paroxysms. Subject to this, the earlier it is given the better. Usually some improvement in the cough is apparent about the fifth or sixth day of treatment. In ordinary dosage it does not kill but merely inhibits *H. pertussis*, and consequently a relapse may occur if the course is terminated much before the twenty-first day of disease. As a rule a ten-day course is indicated or, alternatively, a seven-day course followed by one of five days after a few days' interval. In children of 1 to 4 years administration is not easy owing to the bitter taste of the drug. Infants under 3 months are usually suitable cases for this drug unless the attack is very mild. Dosage is still uncertain. At present the following is suggested: four capsules (1 gm.) a day for infants under 1 year; six capsules from age 1 to 3 years; and eight capsules daily from 3 to 10 years. These amounts should be given in divided doses at six-to eight-hour intervals. In hospital the nurses have little difficulty in giving the drug: they prefer the powder to be disguised in a sweetened or flavoured water vehicle rather than a syrupy one. The child's nose may be gently held if necessary and the draught is then rapidly swallowed.

TREATMENT OF COMPLICATIONS:—For *bronchopneumonia* penicillin is indicated in high dosage, ranging from 100,000 units three-hourly to 500,000 units four-hourly. Severe cases may require to be admitted to hospital owing to the difficulty of treatment and the dangeer of convulsions. The oxygen tent should be employed wherever possible, and chloramphenicol may be required in addition to penicillin in resistant cases.

Convulsions are treated with soluble phenobarbitone intramuscularly or paraldehyde rectally or intramuscularly, but often with disappointing results.

Pulmonary Collapse.—If afebrile, the child should be up and about. Milder cases often clear up without special treatment. Resistant cases should be under the supervision of a specialist. Postural drainage should be encouraged over a Brompton frame, or equivalent double inclined plane, with the head downwards and 12 to 18 in. (30-45 cm.) below the level of the buttocks. Bronchoscopy may be required.

PREVENTION: *Isolation.*—A child with whooping cough should be isolated so far as is possible from young children and especially from infants for at least the first five weeks of the disease.

Immunization.—Pertussis vaccines for prophylaxis alone or combined with diphtheria A.P.T. have been the subject of extensive trials by the Medical Research Council in the last three years. The results will shortly be published. It is known that the incidence and severity of whooping cough in the inoculated cases in these trials were appreciably less than in the controls. Immunization may be attempted at any time after the age of 2 months, but, as in diphtheria immunization, the earlier it is done the greater the need for an early boosting dose. A "booster" should be given after an interval of six to twelve months if immunization has been done within the first year of life.

CHLORAMPHENICOL IN PERTUSSIS

LASSEN AND GRANDJEAN (*Lancet*, 1: 763, 1951) write:

Chloramphenicol was given for 3-5 days to 100 patients with typical whooping cough, most of whom were in the first two or three weeks of the disease.

Repeated nasopharyngeal cultures were examined in every case. Of the 90 patients with positive cultures before treatment 73 became negative within nine days of chloramphenicol being started.

The frequency of the paroxysms fell sharply, and the patient's general condition rapidly improved.

The results obtained with the conventional cough-plate method were compared with those of swab-cultures from the nasopharynx on the same media. The swab-cultures gave a much higher percentage of positive results than the cough-plates, and swab-culture has therefore been adopted as the routine method of diagnosis.

Of 45 strains of *H. pertussis* in which the *in-vitro* resistance to chloramphenicol was estimated, all were found to have a low resistance.

Chloramphenicol seems to be the most effective remedy so far tried in whooping-cough, and it should be used in every case in infancy.

In 93 cases the chloramphenical was administered rectally in suppositories, whereas the 7 older children and adults took it in capsules by mouth.

The smaller dosage which was given to first 19 patients—50 mgm. per kg. of body-weight for only three days—seemed to be less effective than the dosages given to the remaining 81 patients, which were as follows:

Age (yr.)		Per rectum:	Dosage
0-½	...	0·5 gm. plus	0·25 gm. × 4 on first day.
			0·25 gm. × 5 on next 4 days.
½-1	...	0·75 gm. plus	0·5 gm. × 4 on first day.
			0·5 gm. × 5 on next 3 days.
1-6	...	1 gm. plus	0·5 gm. × 4 on first day.
			0·5 gm. × 5 on next 4 days.
		By mouth:	
6-12	...	1·5 gm. plus	0·75 gm. × 4 on first day.
			0·75 gm. × 5 on next 5 days.
12 +	...	3 gm. plus	1·5 gm. × 4 on first day.
			1·5 gm. × 5 on next 5 days.

STREPTOMYCIN IN CHILDHOOD TUBERCULOSIS

BERENBURG AND OTHERS (*J.A.M.A.*, 145: 1044, 1951) write:

The results of treatment with streptomycin in 27 patients with pulmonary tuberculosis are reported. The need for specific chemotherapy in childhood tuberculosis must be carefully assessed in any given patient in the light of previous experience with untreated patients of similar age and with comparable lesions.

Streptomycin is a valuable antibiotic agent in the treatment of pulmonary tuberculosis in children, but it should not be expected to effect a cure in every case. Streptomycin has a particular value in children ill with relatively recent tuberculous pulmonary infections of an exudative or pneumonic type. It is of doubtful value in older children ill with fibrocaseous pulmonary tuberculosis, but may well be useful in younger children ill with this type of disease for a shorter period of time. The optimal duration of treatment and the value of adjunctive therapy are discussed.

PENICILLIN IN PROPHYLAXIS OF OPHTHALMIA NEONATORUM

DAVIDSON AND OTHERS (*J.A.M.A.*, 145: 1053, 1951) write that in 9,241 consecutive newborn treated with penicillin intramuscularly as a prophylaxis against gonorrhœal ophthal-

mia, not a single case of this disease developed. This observation is in keeping with the high gonococcocidal potency of penicillin as reported in the literature.

In an additional series of 4,163 consecutive newborn in whom penicillin ointment, intramuscular injection of penicillin or silver nitrate (1 per cent) was used in weekly rotation, one or more signs of local irritation (redness, œdema or discharge) developed in 10·6 per cent of the penicillin ointment series, in 13·8 per cent of the intramuscular injection of penicillin series and in 48·7 per cent of the silver nitrate series. This observation is in keeping with the general experience that silver nitrate produces chemical conjunctivitis in a high proportion of cases. Provided the silver nitrate has been used in correct concentration, this chemical conjunctivitis does not cause serious or permanent injury to the eye, but a number of cases are on record in which errors were made in the compounding of the silver nitrate, with resultant blindness of the infant. Such tragic errors can be avoided by the use of commercially prepared wax ampules of silver nitrate or by the employment of some other agent.

In the experience of the authors penicillin ointment is the most efficacious, the safest and the least irritating agent for the prophylaxis of gonorrhœal ophthalmia in hospital practice. In addition, it is easy to instil into the conjunctival sac and economical, the cost being about the same as that of the silver nitrate ampules. The recommendation is made that, where necessary, statutes and board of health regulations be changed to permit in hospital practice the instillation of penicillin ointment when the physician prefers it as a prophylaxis against ophthalmia neonatorum.

CURRENT TOPICS

HOW CANCER IS CONTROLLED IN THE UNITED STATES

The following are some of the observations* made by Dr. K. R. Doraiswami, M.A.R.S. (U.S.A.), Madras on the subject while he was in the United States of America in 1950:

Since the mortality on cancer has advanced from seventh to second place (the first being heart disease), the United States Congress has taken an intensive campaign to fight this malady. Babies born in the United States have a life expectancy of 66 years. But 20 per cent of the men and 22 per cent of the women, it is estimated, will have cancer. How many succumb to this depends upon whether better means of cure is found or failing that whether the cancer is detected early enough for successful treatment?

The United States cancer programme is based upon these two points. Its long range plan is to find the cause and improve the methods of prevention or cure. Its immediate plan is to cut short the mortality by earlier diagnosis and prompt treatment. The present cancer programme was launched in 1937 by the National Cancer Institute Act, thus bringing the National Cancer Institute under the Public Health Service and also formed the National Advisory Cancer Council.

What is the National Cancer Institute

This is primarily a research organisation and acts as a division of the National Institute of Health. As we know, the National Institute of Health has made many contributions to the medical world such as discovering the cause of pellagra and the carrier of typhus. This institute was doing cancer research on a small scale even before a separate cancer programme was voted by the United States Congress. Two groups of scientists were doing research in the mid-twenties—one in Harvard Medical School and the other in Public Health Services Laboratory in Washington, under government sponsorship. Their experience gave a trained personnel of about ten cancer specialists around whom the National Cancer Institute was built. Dr. Voegtlin was elected as the first chief of the institute in 1937.

Side by side cancer research was underway in the Universities, hospitals and laboratories but lacked the necessary funds. When the Cancer Institute was established in 1937, it gave assistance to these various institutions on a limited scale.

In 1939, the institute acquired a plot of land kindly donated by Mr. & Mrs. Luke I. Wilson near Bethseda, Maryland, about ten miles from Washington D. C., and constructed its own laboratory and administrative building. Then the War came and interfered with the steady progress of the institute and not until the War was over could the activities be resumed. But the War led to some of the most astounding scientific discoveries like the atom bomb and this encouraged the popular hope that soon another discovery might be made which will result in the cure of cancer. But, for this money was required and money was found. The Congress increased research funds from $490,000 for 1946 to $1,772,000 for 1947. For 1948, the funds were raised to $14,000,000 and in 1949 appropriations totalled $22,000,000. About a third of this went for cancer control and the rest for research.

Composed of six leading experts, each of whom serves a three year period with the Surgeon-General as Chairman, the Council advises the institute and directs the expenditure.

The National Cancer Institute's own work are of two types research and cancer control.

Research

Money alone cannot solve the problem of a cure for cancer. Cancer is as mysterious as life itself. To learn how and why it starts, how it grows and what will stop it, the scientists will have to understand the secret of growth in the individual cell. Very little basic knowledge exists about the laws governing this smallest organised unit of living matter. Yet, considered as a body of accumulated scientific knowledge, we know of more agents that cause cancer than any other disease. It may take many more years of investigation into the unknown aspects of biology before the cause, prevention or cure can be found.

The National Cancer Institute conducts original research of its own and also finances private research through grants-in-aid. It also regularly publishes the latest findings in its own journal. Finally it encourages young and enthusiastic specialists by financing them in intensive cancer research.

It is one of the World's largest organisation consisting of about 250 scientists doing laboratory research in cancer and is situated, as already stated, in Betheseda, Maryland. In addition, the work of the individual scientists is co-ordinated through weekly meetings.

* From a paper read before the South Indian Branch of the I.M.A. on 28-3-1951.

Experiments are conducted in biology where cancer is studied in animals with a view to find out its behaviour in human beings; in endocrinology, in an effort to find out the role of hormones in the causation of cancer and hormonal treatment for cancer of the breast, prostate and uterus; in biophysics to find out the behaviour of cancer cells under the effects of x-rays and radio-active isotopes with the help of high speed ultra-centrifuges and the electron-microscope. Further experiments are conducted in biochemistry to find out what chemical substances are present in cancer and whether they differ from the ones that are present in the normal tissues and discover what chemical reactions cause cancer to grow; in chemotherapy, there is a systematic search for chemical agents that might lead to the development of a chemical treatment of one or more types of cancer in human patients; in cancer pathology where study is made of the cellular structure as it changes from normal to malignant. Then comes the statistics to know how many people at a certain age have cancer; how many recover and how many die and what types and ages are most subject to certain kinds of cancer.

The institute carries on the study on human cancer in the United States Marine Hospital in Baltimore. Special clinical research is conducted in the University of California Medical School and the Garfield Hospital in Washington D. C.

Fellowships

20 research fellowships were granted in 1938. Most of these scientists later became the keymen in the institute itself. Gradually these fellowships were increased to fifty in 1947 and doubled under the expanded programme later on.

These fellowships are available to candidates for a one year period and is renewable. They are awarded to very able men from the states or abroad.

Cancer Control

The United States Government programme against cancer reaches the individual citizens through their respective states. Now most of the states are receiving adequate grants for expansion of their cancer control programmes. These grants differ from that of the National Cancer Institute's research grants in that they are concerned only with diagnosis and treatment. They are made by the cancer control branch of the institute to the official agency in each state that is responsible for the cancer control programme. This is usually the State Health Department. In 1948, about $5,500,000 and in 1949 7½ million dollars were spent on cancer control.

Although the States must submit plans and budgets in order to receive grants, they are free to develop their programme according to local needs. Usually a programme is developed in co-operation with the State Medical Society and the State Cancer Society.

The main aim of the control programme is to reduce the number of cancer deaths by helping the nation's estimated 175,000 practising physicians and their patients make the most effective use of what we already know about cancer.

Aid to the medical profession is given in these ways; through grants up to $25,000 each to medical schools for improving both the quantity and quality of their cancer education, through traineeships to young physicians at $10 per day for graduate clinical study in cancer and through the production of educational materials.

Medical Training

To improve the undergraduate cancer education, the national advisory council invited medical school deans, professors and others interested in medical education to a conference of the National Cancer Institute in November 1946. As a result, the advisory council recommended the following: that medical schools review their cancer teaching and consider adding additional concentrated courses, preferably in the senior year, in which the pathology, physiology and biochemistry of cancer are correlated with a weekly clinic; that they undertake to do cancer research in medical schools as a stimulus to cancer teaching and student interest and that ways be found to financially assist this programme.

Two of the most noted medical schools receiving financial aid in this respect are the Washington University in St. Louis, Missouri and Yale University. There are about 55 other medical schools and 21 dental schools receiving grants.

The institute also provides for traineeships to help graduate physicians take work leading to specialisation in cancer. Traineeships provide up to three years of full time post-graduate work in a hospital with facilities for cancer training, including radiology, surgery and pathology. The trainee may select his own hospital but must be acceptable to that hospital.

Aid to General Practitioner

As the general practitioner comes across only a few cases in a year individually, the cancer control programme arranges with the medical societies, schools and hospitals in keeping physicians better informed on the subject.

One method is through short term refresher courses in which physicians, surgeons, radiologists, pathologists and others can obtain training in modern methods of diagnosis and treatment. Cancer control funds can be used for instruction, meeting places and travel expenses to and from such courses.

Visual materials, such as colour transparencies of cancer tissue, motion pictures and pamphlets are being supplied to medical societies, hospitals and schools. These will make possible the dramatic presentation of up-to-date diagnostic and treatment methods to practising physicians and others.

This type of cancer instruction is also important to dentists who have the opportunity to detect early oral cancers.

Lastly nurses, who have a large responsibility in the care of cancer patients get special training too. Separate courses for both private and public health nurses are arranged and financed.

Cancer Detection Centres

The detection centre has come to be regarded as one of the most essential safeguards against cancer. In such a centre, the physicians and specialists co-operate in periodically examining apparently healthy people for signs of early cancer. These centres have detected cancer in 1½-2 per cent of the apparently healthy persons examined.

Two detection centres were started in 1937-38 and now there are about 200 such centres in operation in the whole of the states. Standards for their operation are prescribed by the American College of Surgeons. Many of them are in the metropolitan areas where they are financed by private organisations.

Diagnostic Clinics

Whereas cancer detection centres serve apparently healthy persons, the diagnostic clinic is equipped to make exhaustive examinations of persons suspected of having cancer. These

clinics operate in conjunction with cancer treatment clinics and are located in general hospitals. To-day clinics are paid accordingly to the amount of service rendered. A fee of $20 from the state Control Agency is allotted to a clinic for each new patient seen and special charges are allowed where the patient is not able to pay for special laboratory examinations. The maximum allowance to a clinic is $4,000 a year for the examination of new patients.

More than 500 such clinics are in operation and the goal is one clinic for 50,000 persons.

The overall objectives of a diagnostic clinic are to provide a consultation service for physicians and dentists in the early diagnosis of cancer. An important function of the clinic is to follow up cancer cases to determine if the patients have been treated and to analyse the results of such treatment. Such procedure permits comparative evaluation of results of various therapies.

In general, the procedure is to admit to the clinic only cases referred by their doctors and dentists. The referring doctor either accompanies the patients to the clinic or presents a written case history to the clinic.

After examination the patient is directed back to his referring physician or dentist and a written report is made giving the diagnosis and recommendations for treatment.

Fully as important as the professional staff of the clinic is a part-time or a full-time medical secretary who maintains proper records of clinical cases, arranges appointments to the clinic and carries on a follow-up of cases of patients diagnosed at the clinic. A nurse is usually assigned by the hospital to assist in the handling of patients on clinic days.

Treatment

There are an estimated 350,000 cancer patients in the states. The National Cancer Institute has a large supply of radium which is loaned free of charge to hospitals. Another important factor is the periodic check-up for recurrence. Laboratory diagnosis is available in tissue diagnostic clinics which also receive grants-in-aid.

Public Education

Lectures are given to the public on the subject and this is mainly carried out by the American Cancer Society.

Cancer Organisations

The biggest and best known private organisation in the field of cancer to-day is the American Cancer Society organised in 1913 by a group of doctors and laymen as the American Society for the control of cancer.

The society's early contribution to the fight against cancer was its educational campaign to convince the public and the medical profession that cancer could be handled if found early enough. In 1937 it enthusiastically supported the entry of the Government into cancer research and in 1945 added research to its own roster of activities.

There are many other private groups carrying on private research. Among these are the Memorial Hospital and the Sloan Kettering Research Institute in the New York City, the Institute for Cancer Research in Philadelphia, the Roscoe B. Jackson Memorial Laboratory in Bar Harbour, Marine, the Rockefeller Institute for Medical Research in New York City, the Barnard Free Skin and Cancer Hospital in St. Louis, the Roswell Park Memorial Hospital in Buffalo, the Chicago Tumour Institute in Chicago, Illinois and the M. D. Anderson Hospital for Cancer Research in Houston, Texas.

A new corner to the field is the atomic energy commission which is spending millions of dollars on cancer research. One of their activities is to supply free isotopes for cancer research in the United States.

General Hospitals—Key-Point of Service

The fate of the cancer patient cannot be decided by a single individual medical man to-day. Group judgment is the wisest course and the management of cancer whether it be surgery, x-rays, radium or radio-active isotopes requires the services of several professional men.

The general hospitals in the States are all well equipped for group judgments which are vital for the diagnosis and treatment. On the staff are found the physician, surgeon, the internist, radiologist, radiotherapist, pathologist, gynæcologist, dermatologist, otolaryngologist, dentist and other specialists, in short all the essential professional men. This form of service is called the cancer service or tumour clinics.

The American College of Surgeons estimate that if there is one such tumour service for 100,000 people, the mortality can be reduced by one half.

The cancer clinic differs from the diagnostic clinic in that while the diagnostic clinic gives the diagnosis and recommends the treatment, the cancer clinic undertakes the entire task—treatment as well as diagnosis.

Many hospitals in spite of limitations are already establishing cancer service and thus providing community service. Group consultations and improved techniques are giving encouraging results.

Whereas the hospitals have enough personnel and equipments, I found that the floor space was wanting everywhere I went. That's why we hear of sky scrapers as the building could only expand upwards and not sideways. Every inch of space was utilised most usefully.

Radiological and clinical laboratories are separately built. There is a Conference Room in which the specialists meet for discussing cases. A projection equipment is provided in this room.

Clinical conferences are an important part of the cancer programme and are of great educational value to the attending, resident and intern staffs. The American College of Surgeons have prescribed minimum standards for these cancer clinics. The staff usually selects one of its members, as the Director of the Clinic. He must be capable of supervising the day to day activities of the clinic and be responsible for them; he must also see that the activities of the clinic are smoothly integrated into the other functions of the hospital. He must be also capable of pacifying some private physicians who consider the clinic as a competitor.

Careful follow-up records of all patients are kept to properly evaluate the results of treatment.

Financial assistance to newly organised clinics for the purchase of apparatus and equipment, can be obtained from many sources—federal, state and local. The cancer control programme of the National Cancer Institute is making funds available to state Cancer Control Agencies. Through these agencies, general hospitals can obtain a great deal of assistance in respect of finances, personnel, both medical and technical. Help can also be secured from the local American Cancer Society.

The fight against cancer is best seen in the United States than anywhere else.

To combat cancer in our country, I would suggest first the formation of an anti-cancer society something akin to

the National Cancer Institute of the United States. This must have the recognition of the Public Health Service and must work in co-ordination. The Surgeon-General should be elected as Chairman. Funds should be collected from generous donors but the lion's share must come from the Government. A committee should be appointed under the Surgeon-General to allocate funds to the various branches of the cancer programme. The programme may be divided into research and cancer control. Under research comes experiments in biology, endocrinology, biophysics, biochemistry, chemotherapy, cancer pathology and specialised clinical research. Fellowships must be instituted to conduct these researches who will later conduct the organisation. The work of the research department can be published in a periodically published journal.

Now under cancer control comes the grants-in-aids to medical schools to improve the undergraduate cancer education, traineeships to young physicians for graduate study in cancer and for educational materials.

As the general practitioner is the keystone for cancer control, post-graduate or refresher courses in cancer must be instituted so that early cases might be promptly recognised by them and sent for treatment. Cancer control funds may be used for instruction and travel expenses for this purpose.

Cancer detection centres should be established in various places and people should be encouraged to visit these places for a check-up. All cancers do not show out in their early stages and a mass survey may lead to detection of early cancer in a small percentage.

Then comes the diagnostic centre where suspected cases can be taken up. Unlike the detection centres which do not require elaborate equipment, these centres can be equipped with radiological and laboratory facilities. A thorough examination is made towards the diagnosis. These centres should be staffed by specialists.

After diagnosis, the patient should be sent for treatment to a cancer service department of a general hospital. This department should be well equipped for giving all types of latest treatment for cancer. The treatment should be chalked out by a group of specialists including a radiotherapist, surgeon, physician, pathologist, gynæcologist, otolaryngologist and others. " No one man should decide the fate of a cancer patient." It is found that group judgment has given the best results.

The detection centres, diagnostic clinics and the cancer service department in a general hospital should be financed from the Cancer Control Funds.

Lastly, the public should be educated on the subject. Lectures should be arranged by the anti-cancer society. This will go a long way in making people cancer-minded.

In conclusion, I will say that I have given you an ambitious plan. Our country is poor and raising funds for such an expensive project will be a stupendous task. But there is no harm in making a small beginning. We medical men must do our duty to the country by fighting sickness and disease prevailing in the country and let me ask the question—" How can we serve the country better."

Finding the cause, prevention or cure of cancer is still a vast problem. The wholehearted co-operation of all individuals and organisations working in the field will lessen the time before the answer is found. The solution to cancer may come from any one of a dozen sources now vigorously being explored but when it will come is a matter of conjecture. Whether it be months, years or decades, it still is in the future.

NOTES AND NEWS

WHO EXPERT COMMITTEE ON CHOLERA MEET IN DELHI

Leading authorities on cholera from six different countries have arrived in Delhi for the first meeting of the Expert Committee on Cholera of the World Health Organisation which is being held in Delhi to find a way of " digging up cholera by the roots."

The international experts will first attend the meeting of the Cholera Advisory Committee of the Indian Council of Medical Research.

Among the subjects to be discussed during the meetings are: The possible role of fish in the preservation and spread of cholera infection; studies in cholera endemicity; the epidemiological importance of cholera carriers; the technique of stool examination; cholera vaccination campaigns; and the importance of a general programme of environmental sanitation.

In its effort " to evolve methods capable of wiping out cholera completely and at not too prohibitive cost " as one expert describes it, the Committee will base its plans on the findings of earlier international Expert Panels and study groups, and on the results of research work carried on in the past three years by the Indian Council of Medical Research and other bodies.

IMPROVEMENT OF PUBLIC HEALTH: UNICEF GRANT TO INDIA

The UNICEF Executive Board, which has just concluded its meetings in Paris, has made a further allocation of 911,000 dollars in support of Government-sponsored health programmes in India, the UNICEF branch office announced at New Delhi on 15-11-1951.

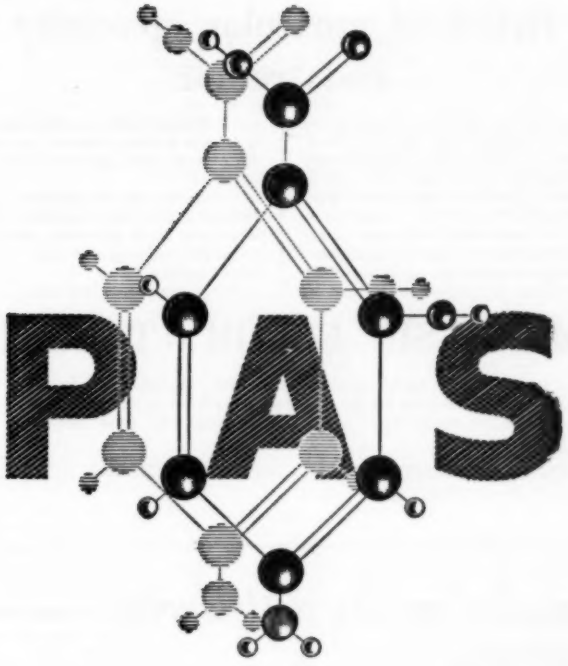
The total allocation is made out as follows:—Development of a mass BCG campaign—$340,000; erection of a DDT plant—$250,000; provision of DDT supplies—$226,000; soap for distribution to health institutions—$25,000; equipment and supplies for maternal and child health work—$70,000 (including $21,000 for equipment and supplies for a hospital at Faridabad).

This new allocation brings the total UNICEF contribution of aid to India to 4,592,000 dollars.

NEED FOR FAMILY PLANNING IN INDIA

The urgent need for concerting methods for family planning in India and for Governments and local bodies to take initiative to educate the public, particularly in rural areas, was stressed in the second annual session of the Mysore branch of the Indian Conference of Social Work on 17-11-1951. The conference discussed only the subject of family planning with a view to focussing public opinion on it. Mr. Gopala Reddy, Finance Minister, Madras, presided over the conference.

Dr. Abraham Stone, Consultant of the World Health Organisation of family planning, outlined his findings on family planning which had eventually been accepted by most Western countries. Family planning, he said, was an ancient practice, but the methods had changed from time to time with the march of civilisation and the growth of knowledge on the subject. Increased food production and maintenance of proper balance between available food and population were the only methods that could avert the impending disaster to humanity to-day.



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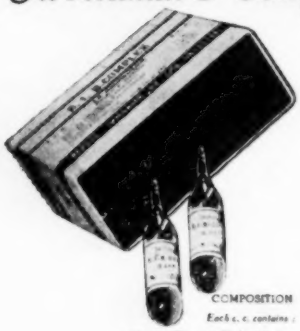

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Each c. c. contains :

Vit. B_1 (Thiamine Hydrochlor)	...	25 mg.
Vit. B_2 (Riboflavin)	...	1 mg.
Vit. B_6 (Pyridoxine Hydrochlor)	...	1 mg.
Nicotinamide	...	50 mg.
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Vitamin B_2 (Riboflavin)	...	2 mg.
Vitamin B_6 (Pyridoxine)	...	1 mg.
Nicotinamide	...	15 mg.
Calcium Pantothenate	...	3 mg.
Sodium Glycerophosphate	...	gr. 4
Potassium Glycerophosphate	...	gr. 4
Calcium Glycerophosphate	...	gr. 4
Strychnine Glycerophosphate	...	gr. $\frac{1}{200}$
Manganese Glycerophosphate	...	gr. $\frac{1}{4}$
Lecithin	...	gr. $\frac{1}{2}$
Alcohol as preservative	...	12% by vol.

PACKING

Issued in Bottle of 6 fl. ounces.

BENGAL IMMUNITY CO., LTD. CALCUTTA 13

Establishment of family planning centres, extension of clinics to teach the use of contraceptives and mass research with a view to evolving birth control methods to suit different peoples of different countries were the needs of to-day. His experiences had shown that the "safe period method" had yielded 90 per cent. success in family limitation, and this method fixed the fertility period which should be avoided a week before and a week after the menstrual period of a woman.

Dr. C. Chandrasekhar, of the U. N. Office for Population Studies, spoke on the rational approach to the problem of family planning in India, and urged that a State-wide family planning programme should be launched and vigorously pursued so that the general basis of our economic structure should be re-orientated and methods suited to each section of society evolved.

Shrimati Sakuntala Paranjpye, of the Family Planning Clinic, Poona, narrated her experiences during the past two decades of work in the field. The need to-day was a band of trained social workers who could spread the knowledge of family planning methods to rural women. Every municipality, she urged, should start family planning clinics.—*Times of India.*

AMENDMENT IN THE BENGAL DRUGS RULES 1946

The following draft of further amendments which in exercise of the power conferred by section 33 of the Drugs Act, 1940 (XXIII of 1940) the Governor proposes to make in the Bengal Drugs Rules, 1946, as subsequently amended, is published, as required by the said section, for the information of all persons likely to be affected thereby. Notice is hereby given that the draft will be taken into consideration on or after the 20th February, 1952 and any objections or suggestions with respect thereto which may be received by the undersigned from any person before that date will be duly considered.

Draft Amendment—In 'Part I—Vaccines' of Schedule E to the said Rules, under the heading '(A) Provisions Applicable to the Production of Bacterial Vaccines,' in paragraph 6 *add* the following clause at the end, namely:—

"(3) In the case of Anti-typhoid-paratyphoid (T.A.B.) Vaccine, the date of expiry of potency shall be indicated on the label of the container. For this purpose, the date of expiry of potency of the vaccine shall be calculated as 18 months from the date of its manufacture."

ISOTOPES FOR MEDICAL USE

Dr. S. S. Bhatnagar, Director, Council of Scientific and Industrial Research who was addressing a distinguished gathering of scientists and physicians at Bengal Immunity Research Institute, Calcutta on the occasion of its fourth anniversary day on 26-11-1951, said that from 1st December next, the Atomic Energy Commission would have the services of a foreign expert in connection with the programme of putting up a pile in near future for making isotopes for medical use.

Stressing the need of extensive research in drugs in this country, Dr. Bhatnagar made a reference to spurious drugs and said that he would very soon publish a list of some drugs manufactured and sold here which were no drugs at all.

Pioneering Work—Recalling the pioneering work done for the drug industry in this country, Dr. Bhatnagar said that drug industry was a necessary industry. He said that he did not wish to discourage drug research, but what he wanted to say was that the human system could not be treated as a test tube. The consequences of a drug should be fully realised by experiments on animals and then statistical averages should be taken before a drug was introduced in the market. It should be realised that drug research was an extensive affair, and it must be paid for if they wanted good results.

Dr. Bhatnagar said that they had already taken over the Kashmir Drug Research Institute and combined it with the Central Drug Research Institute in India. The Institute would remain in Kashmir, but the responsibility of it had been taken by the Government of India.

Modern trend of medical research, Dr. Bhatnagar said, was to shift from plant to animal life. A lot of work was being done in hormones and secretions and the general view to-day was that vegetable matter was after all an intermediary product and it was better to look for the finished product in the animal body for fighting diseases. As a matter of fact, a great deal of extraordinary work had been done in this connection. Dr. Bhatnagar, therefore, pleaded for deeper and vaster study in animal life, in order that fruitful results might be achieved.

Atomic Drugs—Prof. Meghnad Saha said that atomic drugs were now coming into being and it had been seen that many of the isotopes had given wonderful cure. It had not yet been possible to organise a research in this line in India and it was hoped that sooner or later, they would have a pile in India for making isotopes in order that the common man might get drugs at a very cheap price.

Dr. A. C. Ukil, Dr. Hora, Dr. U. P. Basu, among others, also spoke.

AWARD FOR OUTSTANDING RESEARCH IN THE FIELD OF INFERTILITY

The American Society for the Study of Sterility announces the opening of the 1952 contest for the most outstanding contribution to the subject of infertility and sterility. The winner will receive a cash award of one thousand dollars, and the essay will appear on the program of the 1952 meeting of the Society. Essays submitted in this competition must be received not later than March 1, 1952. For full particulars concerning requirements of this competition, address The American Society for the Study of Sterility, 20 Magnolia Terrace, Springfield, Massachusetts.

NEHRU OPENS MEDICAL COLLEGE AT TRIVANDRUM

The Chief Minister, Mr. Kesavan, requesting Sri Nehru to open the college stated that reservation of seats on territorial basis was totally inadequate for the population of nearly 90 lakhs of the state and naturally the doctors qualified in modern medicine were much smaller in the State, *viz*, one for every 12,000. He hoped that women students in the college would be in majority during coming years as the number of the applications showed. He said that at present 20 out of 60 students were women, and mentioned the names of several Malayalees prominently placed in medical profession elsewhere in India including the present Director General of Health Services.

Declaring the college open Sri Nehru said in a short speech that he was impressed with the equipment and site of the college and look of efficiency "from outside." He expressed surprise that the State is standing foremost in education but had no medical college so far. Referring to Chief

Minister's remarks about Malayalees Sri Nehru said amidst laughter that everywhere he was surrounded by them, even in Delhi.

He advised Medical College authorities to maintain high standards and never allow deterioration. Institutions should have social point of view and produce best men. Looking after children, in his opinion, should be the prime concern of medical services.

The college is situated in the midst of picturesque surroundings. Its foundation stone was laid by the Rajpramukh in January, 1950. The college, hospital for women and children with 340 beds, general hospital with 400 beds, school and hostel for nurses, hostel for students, and staff are the chief features of this scheme. The building and equipment, including that of the hospital cost about 130 lakhs. The college building with a magnificent four storeyed structure has one lakh square feet floor space and has laboratories with modern equipment. Government hopes to develop a small township in the college area.

I.M.A., UTTAR PRADESH BRANCH

One of the Local Branches in Uttar Pradesh has offered a prize of Rupees Fifty (Rs. 50/- only) to the member of the Association under Uttar Pradesh Branch who will contribute a paper on Typhoid (Enteric Group) fever.

The paper should reach the Secretary at his office at 43C, Cantonment Road, Lucknow, by 7th March 1952. It should contain the maximum of 15 foolscap sheets, typed at double space and on one side of paper only.

The paper adjudged best shall be read at the U. P. Medical Conference (1952-53) and will be the property of the Indian Medical Association.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors

BLIND RELIEF CAMPS

Sir,—A have myself worked with these Blind Relief Associations for about two and half years. I do not hesitate to acknowledge the pitfalls and handicaps of these camps, which I lay down for the benefit of the public and to draw attention of the Government in this direction.

But for one advantage that of rendering operative treatment to many blind people of the villages at a time, there are many drawbacks. The obvious etiology and the cure are laid down for my brothers. I must stress that only qualified eye surgeons with co-operation of other qualified allopaths and the State Government can do something to overhaul these camps or stop them if they cannot be improved upon.

1. Most of the doctors, in-charge of these Associations, although some are qualified allopaths, are unqualified eye specialists, having gained some training under similar type of time-honoured eye-specialists and who knew little of anatomy, physiology and optics of the eye.

2. An inheritance, they have learnt to operate upon each case in one particular way and would not give up their old teachings for the new methods, nay, they cannot be even moved to improve their methods even if convinced, because they are really not convinced as what was taught to them is a '*guru-mantar*' for them.

3. They can only undertake operations of (*a*) cataract extraction (*b*) iridectomy for leucoma with some success. The iridectomy for chronic glaucoma and the lid operations for entropion which they very often do, are hardly successful even in a few per cent of their so-called successful operations.

4. These doctors on account of their so-called pre-occupations, do not stay in camps during the post-operative period and leave their patients in the hands of their ever-changing apprentice compounders, with the result that these doctors go away with full satisfaction that they had cent per cent success in operations. The compounders in turn are very clever in the tricks of the trade and can fool any educated observer as to the exact results of the operations, for the patients who have remained totally blind since long, will be very pleased with even 5 per cent of normal vision after the operative aid which is rendered to them gratis. These compounders do not even acknowledge the gross failure of vision and loss of eyes due to sepsis or other complications as then they cannot be looked upon by the Association authorities as efficient and run the risk of being replaced.

5. Everybody can well understand how far these attendants can treat the post-operative complications both ophthalmic and medical, which are sometimes a problem even for an eye-specialist and a physician. Such patients who threaten their results on this account and are apt to defame these associations are cunningly sent away to their homes or treated in a manner that these patients run away of their accord and suffer the inevitable consequences of complications.

6. Most of these operated cases are discharged in the last few days of the camp and all the remaining on the day of closing the camp, which is already fixed and the patients still needing care are sent away with one or the other medicine, and some instructions here and there by these attendants, which prevent these poor people from even taking aid of qualified people anywhere till such time when really nothing can be done by anybody.

7. There is no proper supervision of their working. These associations are only source of provision for their own people and for the organisers themselves.

8. These doctors or any body else cannot exactly know the actual results under the circumstances. Patients do not gain at all the normal vision, which is a rarity and practically impossible in these camps on account of all these difficulties.

With a view to overcome these shortcomings and make these camps a real success, I have implemented certain improvements in the working of these camps in my actual practice and have come to the following conclusions:

1. These Associations should be organised by persons with an instinct of sacrificing some of their time. They should have their own source of earning sufficient to make their two ends meet and have no selfish motives or personal interest in running these institutions.

2. Supervision should be in the hands of very well-qualified eye surgeons, who should give surprise visits to these camps and see the results for themselves and not be lured by the operator.

3. The Directors of Medical and Health Services of the provinces should have control over these Associations. They should also collect real results directly from the individuals

operated, through their local medical officers or Patwaries of the villages.

4. The public should as a part of their duty and for their own good, inform the true results (in detail) of these operations after one month to the Director of Medical and Health Services of the province through the local M.O's or Patwaries.

5. Technical staff and the working of these camps; (*a*) The Surgeons holding these camps should be well-qualified for their job and not the type I have described:

(*b*) They should be paid sufficiently so that they do not have to be looking out for back-door practice.

(*c*) They should be bound down to stay in camps throughout the period and not be allowed to leave the patients in the hands of their apprentices.

(*d*) They should keep record of the operative measures and any complications accruing in any case there and then.

(*e*) The iridectomy for chronic glaucoma and such other technical mistakes should be avoided. These cannot be discussed in full in this article of general nature.

(*f*) Aid of tonometer, ophthalmoscope and such other instruments should always be ready at hand.

(*g*) The one-eye cataract glasses which are sold to patients notwithstanding even good vision in the unoperated other eye, should be avoided as a routine for a patient cannot tolerate the great difference of + 10·0 sph. and is apt to have constant headache and slowly lose his sight.

(*h*) The vision of all patients should be taken at the time of discharging not in terms of finger counting but tested on some chart, Snellen's etc.

6. There should be an additional staff, *viz.*, a pathologist, and a physician, who can help the surgeon in not only treating the emergencies and complications but also keep proper and detailed statistics and comparative results. These camps can be the greatest field that can be ever imagined for research work in all the different branches of ophthalmology. The discussion on the proper utilization of this opportunity will be a chapter by itself.

7. The very common eye diseases like xerophthalmia, phlyctenular conjuntivitis, trachoma (granular conjuntivitis) and glaucoma etc., should not be thought lightly of by the eye specialist. He fails from his prime duty of putting an end to these ever-progressing and blindening diseases. There is no fun in our trying to show that we want to help the blind when on the other hand we allow preventable diseases to increase blindness, under our very nose although unconsciously.

8. Definite preventive measures through charts, pamphlets, magic lanterns, if possible through talking movies and lectures, should be adopted to educate the masses. There can be no better opportunity than in these camps.

9. Schools and industrial places should be visited by the eye surgeon and his group, all the students and workers thoroughly examined and given proper advice and treatment.

10. Last, and the most important point is that the Government should enforce certain restrictions of the type suggested to improve upon the working of these Blind Relief Camps and their Associations.

Eye is the most precious of all organs and should not be allowed to be handled by every and any body. I am etc.

Ajitgarh, Amarsar,
30-11-50

J. M. MAKHIJA, D.O.M.S.

VANASPATI: A HUGE HOAX

Sir,—The propaganda carried out by the Vanaspati Manufacturers' Association is so widespread and intensive that even educated people—including doctors—are taken in and they argue in favour of Vanaspati. But any shrewd observer can see that there must be something fishy about a thing which requires so much propaganda. Products like milk and butter and ghee or even oil do not have to be publicised. It is the fake article which needs advertisement.

But let us for a while look at the arguments which a manufacturer of Vanaspati advances in defence of that industry. It is said that more than 22 crores of rupees are invested in the industry; the industry gives direct employment to some 15,000 workers and pays over 3 crores of rupees year in excise duty alone. As to what it pays to Government by way of excise duty, income tax etc., one need only remember that the money comes from the consumers' and not from the manufacturers' pocket. If by prohibiting the manufacture of Vanaspati, the Government loses a few crores of rupees, the nation will gain to that extent, and the nation's gain is also the gain of the Government. As to the number of employees, the same argument could be advanced even by the owner of a factory for explosives. But no sane nation could on that account go on manufacturing explosives even in peace time.

A second argument advanced is that Vanaspati does not even harm the ghee industry. In support of the argument a Government Report on the Marketing of Ghee and other Milk Products in India (1948) is quoted thus: "there has been a *slight increase* in the production of ghee during the last 25 years" (italics mine). Capital is made out of this 'slight increase' and it is said that Vanaspati does not come in the way of pure ghee industry. But if one looks round, one finds that shops where pure ghee is sold are few and far between whereas Vanaspati shops have sprung up everywhere. In the ghee bazar of Ahmedabad, for example, there are hardly three or four shops selling pure ghee, whereas Vanaspati is sold in more than a dozen shops.

A third argument is about adulteration. We, who oppose the manufacturer of Vanaspati say that Vanaspati facilitates the adulteration of pure ghee, and that is one of the many reasons why it should be banned. The advocate of Vanaspati argues. "But, ghee was adulterated long before the advent of Vanaspati. Water is used as an adulterant for milk. Must we then ban water? Mud is used as adulterant for jaggery. Are we to attempt to banish mud?" We admit that ghee was adulterated even before the advent of Vanaspati. But if you go to the Municipal laboratory and make enquiries there, you will be told that to-day at least in all the adulterated samples they find Vanaspati. There is no doubt that Vanaspati facilitates the adulteration of ghee. As to the clever-looking arguments about water and mud my reply is, "We would certainly ban water and banish mud if they were produced in a factory."

It is further argued that the hydrogenated oil industry is not peculiar to India alone; all leading countries of the world use products like margarine as substitute of butter. This is true but there is no comparision between India and western countries. In India, we have been using liquid oil for centuries. Those who cannot buy ghee use some liquid oil. There is no need to hydrogenate this oil. In other countries, there was no substitute for butter (except some animal fat like lard), and so for economic reasons margarine came into the field. But even margarine cannot be compared to Vanaspati. Take, for example the American

brand which is known as oleomargarine and is the most widely used butter substitute. According to McLester (1949) this is "a mixture of several fats that are blended and treated in such a manner as to give the consistency and appearance of the food which it imitates. Its basis is oleo-oil, which is derived from beef fat after the unsuitable fats.....have been removed. To oleo-oil are added a certain amount of neutral lard and occassionally other fats, such as those obtained from the cotton seed, coconut or peanut. There is also added in most instances fish liver oil in amounts sufficient to contribute 15,000 i.u. of vitamin A per pound. *This mixture is churned with milk or true butter* until a homogeneous mass is obtained," (italics mine). Is there any comparison between Vanaspati and oleomargarine?

But you will perhaps get impatient and want to know the scientific status of Vanaspati, because after all, we are scientific men. Let us then examine this problem as nutritionists. We will first turn to the evidence presented by the manufacturers of Vanaspati:—

(1) In one of their broad-sheets they print a glaring headline thus: "A myth exploded" and in slightly smaller types they declare, "Vanaspati is a wholesome food." This is nothing but the language of propaganda. All that the scientists have said is, according to the statement made by the Minister of Food and Agriculture on 8th December 1949 "that there is no deleterious effect produced by Vanaspati as compared with raw or refined oil." Even if it is true that Vanaspati is not deleterious, it does not prove that it is wholesome. Everybody will agree that tea is a harmless drink, but if a tea planter comes forward to say that tea is a wholesome drink, I am sure we would all object to it. Those of you who attended the Second Physical Education Conference held in Ahmedabad in April 1950, will remember what Dr. Radha Krishna Rao of the Haffkine Institute said about Vanaspati. He stated quite definitely that it is in no way superior to the oil from which it is made.

(2) The advocates of Vanaspati claim that Vanaspati can be as easily digested as ghee or groundnut oil and quote in their support the experiment of Roy (1944). According to this observer the digestive co-efficient of ghee made of cow's milk was 99·2 per cent; that of groundnut oil 99·7 per cent and of hydrogenated ground nut oil 99·5 per cent. This experiment was carried out on rats.

Against this I present to you the results of an experiment performed by Professors Basu and Nath (1946) of the Dacca University on *human beings*. According to these investigators the digestive co-efficient of cow-ghee was 97·5 per cent, that of groundnut oil was 99·0 per cent and that of Vanaspati only 89·8 per cent. The article of Roy was published in 1944 whereas that of Basu and Nath was published in 1946. They had also mentioned "Dalda" by name.

(3) It is further stated that Vanaspati is as easily absorbed as groundnut oil, and in support is quoted the experiment of Nhavi and Patwardhan (1946) performed on human beings. These workers had used "partially hydrogenated groundnut oil" in their experiment. It is interesting to read their remarks on this point. They state: "It is not suggested, however, that a completely hydrogenated product which must have a higher melting point, would be absorbed at the same rate as the original oil."

How deceptive the propaganda for Vanaspati can be, and how easily people are taken in is best illustrated by an article of Lt.-Col. Amirchand (one of our past presidents), which he wrote in a newspaper of Delhi.* I shrewdly suspect that all the arguments which our friend has advanced in favour of Vanaspati are borrowed from a circular of the Vanaspati Manufacturers' Association. Even the scientists who have declared that Vanaspati is not deleterious have never ventured to state that Vanaspati is as good as ghee, but Col. Amirchand goes one better and states that as far as energy and fat food value are concerned, those of Vanaspati are "in no way inferior to that of ghee."

I must now give you my reasons for opposing the use and manufacture of Vanaspati.

(1) First and foremost, the process of hydrogenation raises the melting point of the oil which is hydrogenated. The experiments of Langworthy made more than thirty years ago showed conclusively that fats of low melting point are more completely assimilated than those with a higher melting point. A similar conclusion was reached in the recent studies of Mattill and Higgins (1945). At the Science Congress held in Bangalore in 1946 the question of Vanaspati was discussed and everyone agreed (even the advocates of Vanaspati) that fats with a melting point higher than the temperature of the human body (*viz.*, 37°C) are poorly absorbed. Extensive experiments on rats were carried out at the Imperial Dairy Research Institute in Bangalore. According to Dastur (1946) who sponsored these experiments, "The most significant result of these studies was that hydrogenation greatly diminishes the rate of absorption of all the oils studied and the rate decreases as the melting point rises."

Against this, it will be argued that the Government have now laid it down that the melting point of Vanaspati shall be 37°C and that this law came into effect after (or because of) the Science Congress held at Bangalore. It may further be argued that recent experiments on rats made by Misra and Patwardhan (1948) at Coonoor show that "there is no appreciable decrease in digestibility of hydrogenated fats melting between 29·5°C and 49°C."

(2) But let me give you the details of an experiment made also at Coonoor, and under Dr. Patwardhan, but on human beings. In this experiment, it was demonstrated that the digestibility co-efficient of groundnut oil was 94·03 per cent, of Vanaspati with a m.p. of 37°C 93·73 and of Vanaspati with a m.p. of 41°C. 89·89 per cent. These figures conclusively show that liquid oil is in no way inferior but rather slightly superior to Vanaspati even when the m.p. is 37°C. Further, Vanaspati interferes with the phosphorus and calcium metabolism in our body. From 840 mgm. of phosphorus provided in the diet, 127 mgm. were absorbed into the body with groundnut oil; 142 mgm. with 37°C m.p. Vanaspati and only 52 mgm. when the m.p. of Vanaspati was 41°C. Vanaspati acts still more deleteriously on calcium metabolism. Out of 604 mgm. of calcium of the diet 70 mgm. were absorbed with groundnut oil; 22 mgm. with Vanaspati of 37°C m.p. and none with Vanaspati of 41°C. m.p. Not only so, but there was a negative balance of 42 mgm. with the latter brand of Vanaspati—that is to say that calcium is washed out of the body. If you remember how important an element is calcium in our diet and how poor the usual vegetarian diet (particularly that of the rice eater) in calcium, you will at once agree that *Vanaspati is certainly deleterious to health.*

But the advocate of Vanaspati will smile superiorly and contemptuously point out that the Government have fixed

* *Hindusthan Times* of 26-10-1950.

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37°C as the limit for the melting point of Vanaspati, whereas the negative calcium balance occurs only with Vanaspati of a m.p. of 41°C. True, but the Government allows a variation of 2°C. which means that Vanaspati with a m.p. of 39°C is admissible. We must also remember that Government have not appointed a censor at every factory who takes the m.p. of all the Vanaspati produced. Further, if 42 mgm. of calcium is washed out of the body when the m.p. is 41°C, it is reasonable to assume that at least 10 mgm. will be washed out when the m.p. is 39°C. Let us, however, assume that no calcium will be washed out of the body. But surely and certainly, none will be absorbed and no one can doubt that more calcium is absorbed into the body with groundnut oil than with Vanaspati even when the m.p. is 37°C.

These results on calcium and phosphorus metabolism have also been reported in rats by Kehar (1947). He has further shown that the biological value of protein decreases when given with Dalda or Kotogem.

(3) Experiment in animals have shown that certain fatty acids are essential to the organism. Linoleic and linolenic acids are such essential substances. When oil is hydrogenated, these acids are destroyed partly or wholly. This fact is confirmed by the 1946-47 Report of the Research Organization of the American Government. This report states, "The process of hydrogenation destroys linoleic acid (and other unsaturated fatty acids) to a large degree and thus removes vitally needed food substances from natural fats." This report also indicates that all hydrogenated fats, whether of animal or vegetable origin are somewhat harder to digest than unhydrogenated fats (Ref: Editorial, *Health*, Oct. 1950).

(4) Lastly, we come to the vitamins. As a rule vegetable oils do not contain vitamins A, B, C or D. But they are fairly good sources of vitamin E. No work has been done in India on the vitamin E content of foodstuffs, but such work has been recently undertaken in America. This work shows that coconut oil contains 8·3 mgm. of vitamin E per 100 mgm. but when coconut oil is hydrogenated, the amount of vitamin E is reduced to 3·0 mgm. only. In other words, the process of hydrogenation destroys vitamin E. This is one more reason for opposing the hydrogenation of oil in our country. (Ref: *Journal of Nutrition*, March 1950, pp. 367-381) and here, I wish to sound a note of warning to those who are interested in the study of nutrition. The advocates of Vanaspati quote the experiment of Datta (1945) of Haffkine Institute in proof of their claim that "hydrogenated vegetable fat is as good as butter fat in supporting growth." It is also possible to get from the literature other similar experiments. It is my contention that these experiments, though technically perfect, do not represent the human situation. In Datta's experiment, for example, all the rats were given "a supplement of two drops codliver oil." Now, when human beings use Vanaspati, they do not say to themselves, "Vanaspati does not contain vitamins A and D. Let me therefore take some cod-liver oil with Vanaspati." We do not compare Vanaspati and ghee only as chemical substances but as foodstuffs. If one batch of rats is given Vanaspati as fat the other should be given ghee and to neither of these should cod-liver oil be given. It may be argued that in Dutta's experiment, cod-liver oil was given to all the rats. But in my humble opinion, that did mask the deleterious effect of Vanaspati. If we accept Datta's method, we are going back to the nineteenth century when the chemists believed that cod-liver oil was no better than corn oil because both were fats, and chemically (as they then thought) all fats are alike. But no; we accept the results of McCollum and Davis, and Osberne and Mendel (though their experiments date back to 1913) who showed that "rats could not grow or remain in good health when lard, olive oil or almond oil provided the sole source of fat, whereas their condition was much improved when these fats were replaced by butter fat, cod-liver oil, or egg-yolk fat." (McCollum, 1939). Hathaway and Davis (1935) reported that rats on butter diet lived and made consistent gains in weight whereas all the rats on oleomargarine lost weight and of the total of 75 rats only 5 survived the 8 weeks feeding period. Additional feeding tests involving a total of 370 rats showed similar results. If these are the results of oleomargarine which contains both animal and vegetable fats and is said to be "equal to butter in vitamin A content" you can understand how absurd it is to claim that Vanaspati supports as good growth as butter! All that the recent experiments show is that if all the defects of Vanaspati are remedied, it can be as good as butter.

The conclusion, then, that we arrive at is this: Liquid oil is a good substitute for ghee for those who cannot afford ghee. Oil should not be hydrogenated (1) because the process of hydrogenation raises the melting point and makes it less digestible; (2) because it hinders the metabolism of calcium; (3) because it destroys partly or wholly the essential fatty acids; (4) because it destroys part of vitamin E and (5) because it facilitates the adulteration of ghee and (6) makes the original oil more costly.

To those who favour the half-way measure of colouring Vanaspati I have this warning to give. The vested interests of Vanaspati are so powerful that they will try all their might to prevent such a measure. Let me quote to you from a circular issued by the Controller of Vegetable Oil Products to the Provincial Governments and Minor Administrations. Says this high Government Official,"..... I am to request that your Government may reconsider if any legislation aiming at compulsory colouration of Vanaspati is essential as it will result merely in destroying the psychological appeal of Vanaspati to the public without making detection of adulteration any more effective. I would bring to your notice that as yet no colourising material which would not affect the keeping quality of Vanaspati, would not be injurious to health, would not be easily eliminated by heating or by chemical process, would be non-toxic and which would not be objectionable to sight, smell or taste, has yet been discovered." In other words, this Officer of the Central Government is doing his best to prevent the compulsory colourization of Vanaspati. One wonders whether these words come from a Government servant or from a servant of Vanaspati Manufacturers' Association.

Let us then wake up and not be content with the half-way house measure of colouring Vanaspati but let us ask for a complete ban on the hydrogenation of all edible oils. Let us all support the resolution passed by the A.-I.C.C. at Ahmedabad and unanimously ask for the banishment of all Vanaspati.* I am etc.

Ahmedabad,
5-4-51.

K. A. SHAH,

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HEALTH CONSCIOUSNESS IN THE PUBLIC

Sir,—It has been repeatedly declaimed that the health of the people of India is deplorably below par and that strenuous efforts must be made to mend the same. The Central and State Governments as well as the enlightened class are fully aware of the state of things. There is no doubt that some work is being done by the authorities and benevolent voluntary associations to improve the health of the people. But it must be admitted that health consciousness in the public is an essential factor to raise the hygienic and sanitary living of the masses. No movement will succeed as long as it is not supported by the people themselves. Constant propaganda on sound and practical basis will be a great help in creating this consciousness and accelerating the progress of national health.

This important and vital question demands the closest attention and consideration at the hands of the Indian Medical Association. It must be emphasised that local branches should take up the question in hand and direct their members to do propaganda all the year round in their localities. Members in their daily practice come in close contact with the local residents and wield great influence in the families. They should advise patients and their relatives to take preventive measures and cultivate clean habits. Mere administration of medicines to cure patients should not be supposed to be the only duty of medical practitioners. Advice on preventive measures is as important as that on the curative side of a practice. Importance of clean habits and hygienic living must be repeatedly impressed on the public mind.

Enthusiastic members should deliver lectures in different parts of their towns at intervals—they should arrange what we call "dustbin" lectures in co-operation with local civic authorities. Such lectures with visual educative means are always better attended than mere verbal talks. Film and magic lantern lectures are attractive to young growing people and female folk to whom we should always appeal.

Local branches should form sub-committees to look into the matter. Sub-committees can be asked to give a report of their activities every six months to the general body. Committees and six-monthly reports should be statutory in the local constitution of the branch. In this way on some members will be laid direct responsibility to carry out the necessary propaganda.

Local bodies (municipalities, local boards, and panchayats) should be advised to organise a permanent museum where models, charts, pictures etc., on health matters would be kept to interest the public. Such propaganda should be especially directed in the backward classes and to slums in urban areas.

Restaurants and sweetmeat shops have assumed great importance on account of the great increase in patronage extended to them. They are hotbeds of all sorts of diseases. Members should take interest in the sanitation and cleanliness of these hives. People should be advised to bring pressure on restauranteurs and vendors to keep their establishments clean and decent. Local authorities should take doctors and customers into their confidence and with their aid they should take vigorous action against offenders. In case of offences against sanitation and public health, legal procedure should be simple and speedy; otherwise it becomes very difficult for the health authorities to proceed against offenders and bring them to book.

Urban areas have different agencies to impart health education, such as local self-government bodies, teaching institutions, practising doctors and their associations. In villages no such agencies exist. Here subsidised medical practitioners must be asked to take keen interest in preventive measures of diseases and in giving lectures to villagers on health matters. They must help the mobile sanitary staff whenever they visit their localities.

School-going children are impressionable and should be tackled early and well. They must be made to understand the importance and the ways of sanitary and hygienic living. I am sure in the long run succeeding generations will go on improving in this way. State Governments should be induced to appoint members of the Indian Medical Association on school text-book committees, so that they might help introduce the right type of lessons to impart education on health and hygiene to school-going children.

Legislation alone on sanitary delinquency will not be of use, unless health consciousness is created in the population. I am etc.

Nasik,
6-6-1951. P. S. GUPTE, M.B.B.S.

UNILATERAL EXOPHTHALMOS

Sir,—In the May 1951 issue of your journal in the Practitioner's Corner, there appeared an article entitled: "Unilateral Exophthalmos—Its etiology with special reference to malignant growth."

The author in reporting his case mentions of an evanascent swelling in the skull, which statement is rather vague and probably means that the swelling was in the scalp. From the description of the x-ray and physician's reports and to some extent from the rather incomplete description of the proptosis and the pathologist's report, I deduce, it is a case of sarcoidosis.

Proptosis due to Boeck's sarcoid is not so rare. The granulomatous uveitis is a characteristic feature associated with changes in lungs and long bones. The disease is progressive with irregular periods of abeyance and remissions—sometimes with restoration and recovery—other times, a devastating uveitis, destroying visual function.

It would be illuminating if one knew of the presence of either conjunctival or skin nodules. A biopsy of a lymph gland would demonstrate the presence of Schaumann bodies present in nearly 40 per cent of cases. The deviation of blood calcium level from normal, as well as an increased E.S.R. and a relative increase in albumen and globulin, would indicate in conjunction with other findings, the presence of sarcoid.

I hope by now the histopathological examination of the eye has been made; I would be grateful if I could know the pathologist's report. I am etc.

11-11-51
Nawapura St., N. T. MASCATI, D.O.M.S.
Surat.

REVIEWS

MODERN TREATMENT YEAR BOOK 1951, edited by Sir Cecil Wakely, K.B.E., C.B., D.S.C. Pp. viii & 356 with 21 plates, Price 17s. 6d. net. Published by Bailliere Tindall and Cox, 7 and 8, Henrietta Street, London, W. C. 2.

This is a year book presented by the Medical Press containing a variety of articles most of which are interesting and valuable to the general practitioner. Special emphasis has been paid to the amount of caution to be exercised over the use of antibiotics which have revolutionised the treatment of many conditions. Practically the whole field of medicine and surgery has been discussed in the volume. Special stress has been made on diagnosis, and modern methods of treatment have also been discussed. Virtually all the modern progress in diagnosis and treatment have been incorporated in the book. We commend the book to the busy general practitioners who would find in this book a mine of informations to help them in their daily professional work.

BENGAL MEDICAL DIRECTORY 1951, edited by Dr. P. K. Guha and published under the auspices of Indian Medical Association, Bengal Provincial Branch by Messrs. Hopewell & Co., 31 Beckbagan Row, Calcutta 17, Price Rs. 12/8/-.

The book contains a variety of materials meant for the medical profession and others interested in the development of medicine in this country. "History of modern scientific medicine in India," contains a lot of information, but in our opinion it could have been more detailed and elaborate in the interest of doctors and medical students. Details of medical educational institutions, medical council and other learned societies of different codes of medical ethics, reports of medical relief of the West Bengal Government, Corporation of Calcutta, statistical reports about the province, Drugs Act provisions, Resolutions passed by the I.M.A Conferences and list of doctors who are members of the I.M.A. in Bengal with names of branches are incorporated in the book. Barring a few misprints the book is expected to be a useful guide for ready reference not only to doctors but manufacturers and others also.

YOUR QUESTIONS

Correspondents should give their names and addresses (not necessarily for publication) and include all relevant details in the questions which should be typed. Questions of medical interest only will be acceptable for this section. They should be sent in a separate envelope marked "Your Questions." Questions of general interest will be published in preference. Lack of space precludes answering all the questions received.

Q. 1. Is it possible to get a positive sugar reaction in the urine though there is no hyperglycæmia?

2. In what condition presence of yeast cells in excess may be found in urine? Can it give a positive reaction to sugar in urine?

Ans. 1. Glycosuria may occur with a normal level of blood sugar in renal glycosuria. The condition is associated with a lowering of the renal threshold for sugar. In late stage of pregnancy and after parturition lactosuria is often present. Lactose also gives a positive copper-reduction test. In these cases the blood sugar is normal unless the patient is a subject of diabetes.

2. "Excess of yeast cells" in the urine is usually indicative of a contamination. Urine, especially that of a diabetic person may be found to contain yeast, if left in an open container. Monilia infection of the urinary tract is rare. In the female, if monilia vaginitis is present, yeast cells may be found in the urine unless a catheter specimen is tested. For the same reason, female diabetic subjects often show the presence of yeast cells in the urine. Yeast does not give a positive sugar reaction. On the contrary, heavy contamination with yeast may remove the sugar already present in the urine.

Q. 3. A patient aged 13 has moist "seborrhœic dermatitis of scalp where scales in abundance come out on scratching the scalp. Hydrargyri per. chlor, Bepanthen have been tried in lotion—no use. Kindly advise treatment.

Ans. 3. *General treatment* consists of improving the endocrine imbalance by some tonic like the 'Incretone' (Carnick) taken regularly twice or thrice daily. 'Ethidol'—Schering (Ethinyl œstradiol) is worth trying in the following doses, 0·001 mgm. once daily for 15 days, then stop for 7 days, then repeat for another 15 days. The third course and afterwards—0·001 mgm. once daily every alternate week. (Remember the dose is 0·001 mgm.). Careful watch should be kept for any change in the physical and/or mental condition of the boy and ethidol should be stopped or regulated accordingly.

Local treatment should be as follows: 1. Shave the scalp and keep the hair short (very short) throughout the course of treatment which is likely to last 6 *months*. 2. In the evening rub the Hyd. Oleate Ointment (A) on the scalp for five minutes and then tie a handkerchief or a piece of linen over the scalp for the night. 3. In the morning-remove the ointment with olive oil and wash the scalp with the shampoo (B). First wash off the soap with warm water and then use cold water for the bath. 4. After bath rub the lotion (C) twice daily (morning and afternoon). The strength of the ointment, shampoo or the lotion may be reduced if found necessary.

A. Ointment

Hydrarg. Oleate 10%	...	...	dr. 1
Acid Salicylic	...	...	gr. 10
Glycerine	...	...	dr. 1
Oil Olive	...	...	dr. 3
Eucerinum Anhydrous ad.	...	...	oz. 1

Ft. Ung. to rub.

B. Shampoo

Soft Soap	...	...	oz. 1
Tr. of Lavender	...	...	oz. ½
Spirit of Ether	...	...	oz. 1
Rectified Spirit	...	...	oz. 1½

Ft. Soap for Shampoo.

C. Lotion

Resorcinol	...	...	dr. 1
Tr. Cantharidine	...	...	m. 15
Oil Ricini (Allenbury)	...	...	m. 15
Spirit of Ether	...	...	dr. 1
Rectified Spirit	...	...	dr. 1
Rose water upto	...	...	oz. 1

Ft. Lotion to rub.

The above treatment should be carried out for six months with changes mentioned above before any other method is tried.

NON-RECEIPT OF JOURNALS

Subscribers and members of the various branches of the Indian Medical Association are requested to note that all communications about non-receipt of the Journal of Indian Medical Association should be addressed to the undersigned.

In case the address is not correctly recorded or there has been a change in the address, an intimation should be sent forthwith.

For despatch of Journals the residential address is preferable and not the hospital, college, chamber or dispensary address in which case the Journals are likely to be lost.

Be it noted that Journals are despatched in the last week of the previous month. In case of non-receipt of Journals *within the first fortnight of the month* it is requested that an intimation be sent to the undersigned within the months. Further, it is also desirable that a written complaint be lodged at the local post office and the reply received there to be also sent to the undersigned to take the matter up.

Co-operation to this effect is earnestly solicited.

J. MOJUMDAR,
Hony. Secretary,
Journal of the Indian Medical Association.

1-1-52.

XXVIII ALL-INDIA MEDICAL CONFERENCE

Important Information for Delegates and Visitors to the Conference

Railway connections.—Ahmedabad is three hundred miles west of Bombay and five hundred miles from Delhi by metre gauge.

For all delegates from South beyond Bombay the most convenient trains for Ahmedabad are—Janata Express (Class III only and Gujarat Mail leaving Bombay Central at 19-50 and 20-20 hours reaching Ahmedabad at 6-0 and 6-45 A.M. respectively, hence reservations should be secured by these trains only.

For delegates and visitors from Bengal, Bihar, U.P., Punjab, Delhi etc., the convenient train is Delhi Mail, metre-gauge, leaving Delhi at about 21-30 hours and reaching Ahmedabad at 21-0 hours next day. This is the shortest journey from Delhi hence reservations should be secured from Delhi to Ahmedabad by the Delhi Mail (metre gauge). Besides, there is Delhi Express (metre gauge) leaving Delhi at about 7-30 A.M. reaching Ahmedabad next day at 12-0 A.M. Frontier Mail and Meerut Express (broad gauge) running between Delhi and Bombay *via* Baroda can be availed of but with a change at Baroda during night hours which is inconvenient.

For delegates and visitors from C.P. and C.I., they can travel from Bhuvasal to Surat by Tapti Valley Railway leaving Bhuvasal at about 23-0 hours reaching Surat at about 11-0 A.M. and by Saurashtra Express from Surat at 13-0 hours reaching Ahmedabad at 20-0 hours. They can also travel *via* Indore and Ratlam and change at Baroda for Ahmedabad. They can also travel *via* Bombay which is a longer route.

For Saurashtra and Gujarat members there are several trains reaching Ahmedabad during morning and evening hours.

For Air travel there is regular service from Delhi to Ahmedabad *via* Bombay:—1. Tata Air-Plane leaves Bombay (Santa Cruz air port) at about 7-30 A.M. reaching Ahmedabad at 9-30 A.M. At present there is no direct service from Delhi.

Arrangement for Reception at the Station.—There will be a Reception Camp at the Ahmedabad station and also at Ellis-Bridge station, where volunteers will be available for all trains on both the broad and metre gauge platforms.

Transport.—At the station platform volunteers will take charge of the kits against a receipt and arrange for transport. At the camp delegates and visitors will recover there kits on production of the receipt.

Camp Arrangement.—The camp is situated in the premises of Sheth C. N. Vidyalaya, Ellis-Bridge, where all programmes and deliberations of the Conference are fixed. For delegates and visitors from outside Gujarat and Saurashtra, arrangements for lodging and boarding will be made at the camp. Delegates and visitors from Gujarat and Saurashtra are requested to make their own lodging arrangements if possible, failing which, the Reception Committee will do the needful on timely intimation. There will be separate arrangement for lady doctors and doctors with families on previous intimation.

Food Arrangement.—There will be a common camp kitchen where all delegates and visitors will be served on tickets, tea, breakfast, lunch and dinner for both vegetarian and non-vegetarian diet but owing to strict rationing in the city, delegates and visitors must inform the Reception Committee before 15th. January, '52 whether they will dine at the camp or not. They are also requested to bring proper warm clothing and complete bedding.

Railway Concessions.—Concession tickets for all classes will be available from railway stations on production of the certificate from the Organising Secretary and the charges will be at the rate of single fare double journey.

Lastly, delegates and visitors are requested to intimate the Organising Secretary before 15th. January, '52 about:—

(1) Their arrival and departure time and train. (2) Their requirements about boarding and lodging. (3) Whether vegetarian or non-vegetarian. (4) Their concession ticket certificates.

Branch secretaries are requested to send the list of delegates and visitors immediately. For any further information please write to the Organising Secretary, 28th. All-India Medical Conference, Prarthana Samaj Hall, Raikhad, Ahmedabad-1. Telegraphic address—Medicon Ahmedabad.

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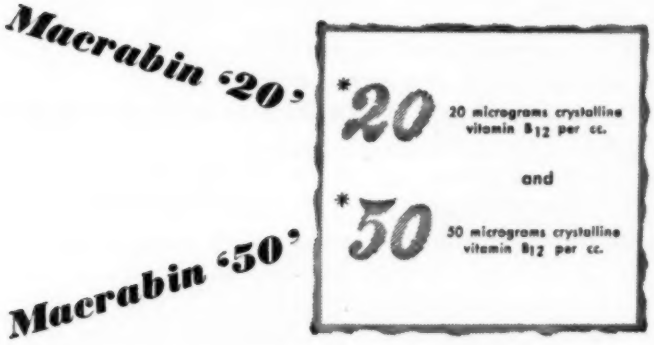

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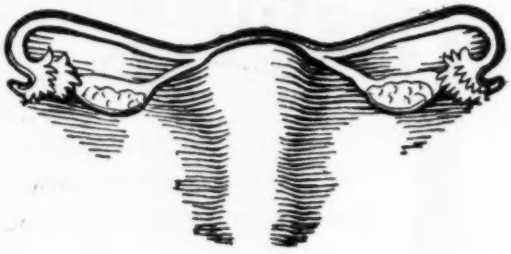

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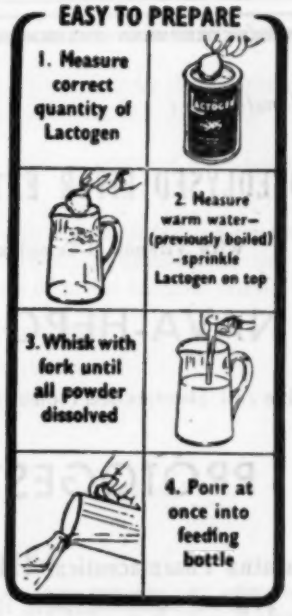
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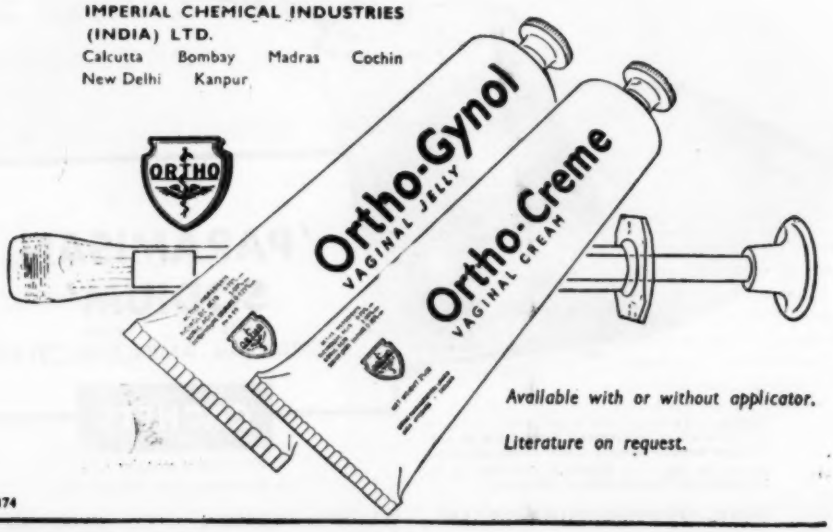

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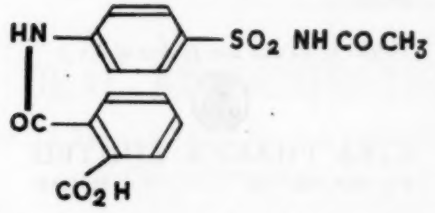

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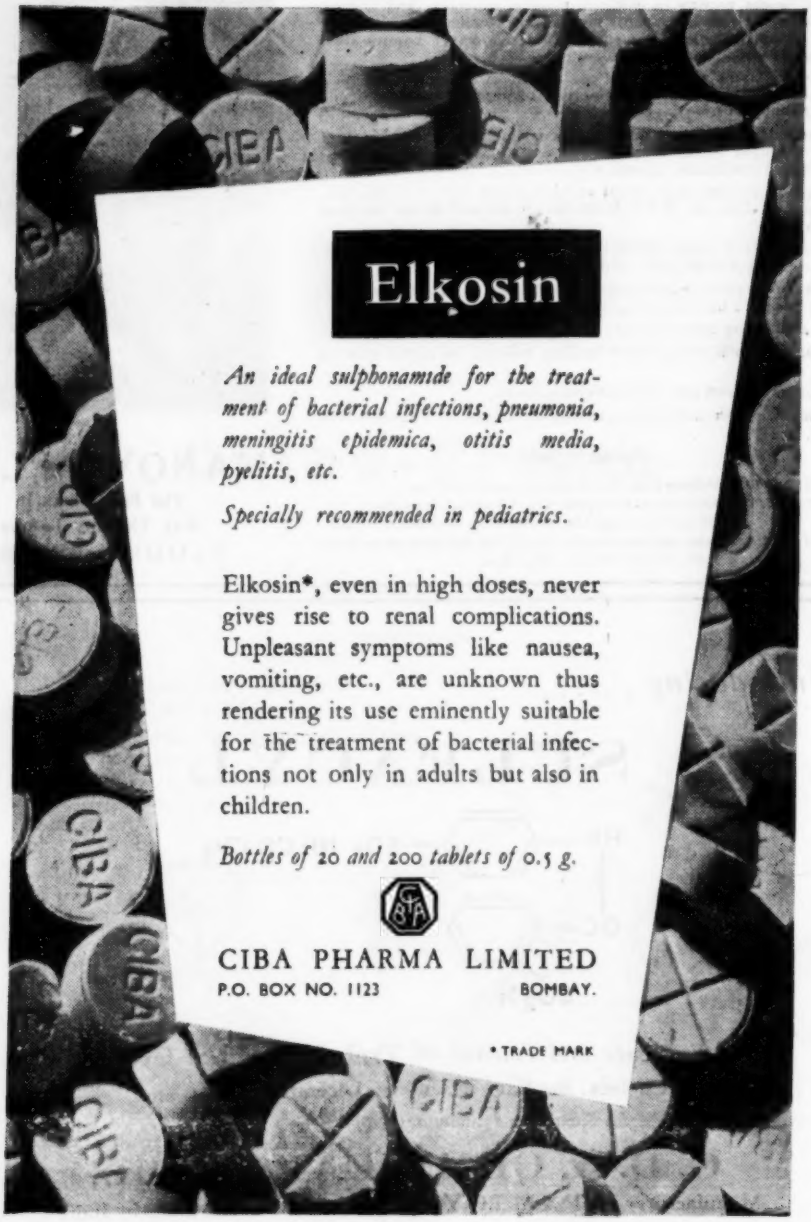
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